# A COMPARATIVE STUDY BETWEEN THREE METHODS FOR WEANING OF TRAUMATIC HEAD INJURY PATIENTS FROM MECHANICAL VENTILATION

*Thesis*

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M. D. Degree in anesthesia and critical care

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"قالوا سبحانك لا علم لنا
إلا ما علمتنا إنك أنت
العليم الحكيم"

صدق الله العظيم

آية ٣٢"– "سورة البقرة

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# AIM OF THE WORK

This prospective observational study is aiming at how we can best determine when a patient is ready to be liberated from mechanical ventilation, and how we can identify modifiable factors with planning and implementation of corrective actions aiming at decreasing the failure rate of extubation, decreasing the mortality rate, and if we can identify the best way of weaning head injured patients from mechanical ventilation comparing three methods which are: synchronized intermittent mandatory ventilation (SIMV), pressure support ventilation (PSV), spontaneous breathing trial (SBT) in order to improve our ICU outcomes.

# HISTORY OF MECHANICAL VENTILATION

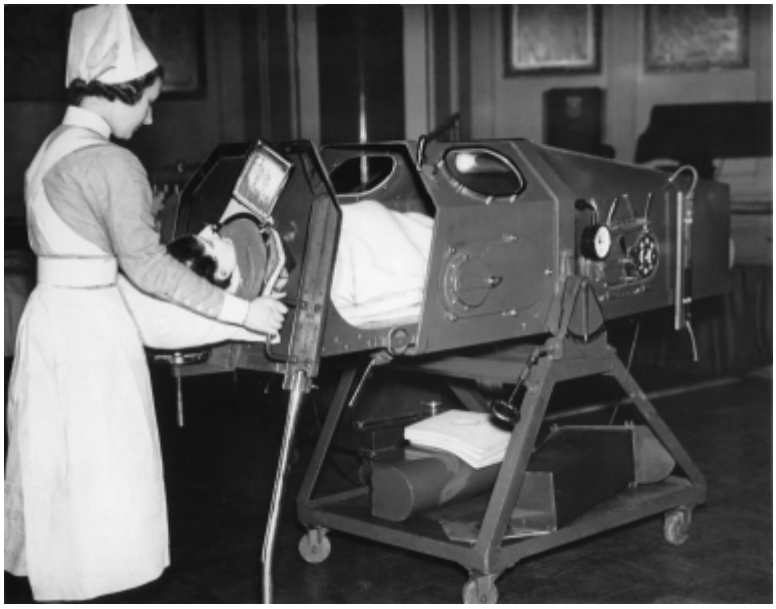
Ancient writings by the Egyptians and Greeks described theories of respiration. In the Old testament there is a mention of Prophet Elisha inducing pressure breathing from his mouth into the mouth of a child who was dying (Kings 4:34-35). Hippocrates (460-375 BC) wrote the first description of endotracheal intubation his book –'Treatise on Air' "One should introduce a cannula into the trachea along the jaw bone so that air can be drawn into the lungs".

Aracelsus (1493-1541) used 'Fire Bellows' connected to a tube inserted into patient's mouth as a device for assisted ventilation. This was the first study (1550) which credited him with the first form of mechanical ventilation. Vesalius (1543) performed ventilation via a tracheostomy in a pig. Hook (1667) used bellows via a tracheostomy in a dog. John Fothergill in 1744 reported a successful case of 'mouth to mouth' resuscitation. John Hunter developed double bellows for resuscitation in 1775 - one for blowing air in and the other for drawing bad air out. Dragger Medical designed an artificial breathing device "Dragger Pulmoter" in 1911 that was used by fire and police units ***(Colice, 2006)***.

From the mid 1800-1900s a large number of devices were invented that applied negative pressure around the body or thoracic cavity – these devices became known as negative pressure ventilators or 'iron lungs'. Two successful designs became popular; in one, the body of the patient was enclosed in an iron box or cylinder and the patient's head protruded out of the end (figure 1). The second design was a box or shell that fitted over the thoracic area only (chest cuirass) (figure 2). Patients with chronic paralytic disorders were successfully ventilated on these cuirass ventilators at home for 25-30 years.

In Copenhagen between July and December of 1952, 2722 patients with poliomyelitis were treated in the Community Disease Hospital of which 315 patient required ventilator support.

Many principles of IPPV were defined during that time –including the use of cuffed tubes, periodic sigh breaths and weaning by reduction of assisted breathing.



**(Figure 1)** A Polio Patient being placed in an Iron Lung, 1938. ***(Tobin, 1994)***

**(Figure 2)** Chest cuirass ***(Tobin, 1994)***

Towards the end of the epidemic a few positive pressure ventilators were invented (the Engstrom, Lundi and the Bang) which became popularly known as mechanical students.

After polio epidemics, the 1960's became an era of respiratory intensive care. Positive pressure ventilation with use of an artificial airway replaced the bulky and cumbersome negative pressure technology of respiratory support.

Two types of ventilators and two modes of mechanical ventilation evolved during this period; the first type of ventilator was pressure cycled (PCV) ***(Tobin, 1994)***.

Two ventilators were commonly used for PCV in the 1960's and 1970's; the Bird Mark 7 and the Bennet PR2. The second type of ventilator that evolved from a historical perspective is the volume cycled ventilator (VCV). The first fluidic ventilator utilizing moving streams of liquid or gas for sensing, logic, amplification and controls was designed for the US army in 1964 by Barila and the first commercial versatile fluidic ventilator "Hamilton standard PAD" appeared in 1970. The term 'weaning' was used to explain various techniques to test the quality of patient's spontaneous ventilation before extubation.

A mechanical change of substantial importance in the late 1960's and early 1970's that shaped the present era was the introduction of Positive End Expiratory Pressure (PEEP). Two modes of ventilation Assisted Ventilation (AV) and Controlled Mechanical Ventilation (CMV) came together in a single piece of equipment and the modern era of multiple choice respiratory support was born. The introduction of Intermittent Mandatory Ventilation (IMV) permitted spontaneous respiration in the midst of substantial respiratory failure which paved the way for a means of weaning i.e. Synchronized Intermittent Mandatory Ventilation (SIMV). Pressure Support Ventilation (PSV) proved to be an addition to IMV that facilitated spontaneously breathing patients ***(Chamberlain, 2003)***.

# FUNDAMENTALS OF MECHANICAL VENTILATION

The basic purpose of mechanical ventilation is to support patients whose respiratory systems have failed until adequate function returns. Reversal of acute, severe hypoxemia or respiratory acidosis with mechanical ventilation can be life- saving ***(Tobin, 1994)***.

Mechanical ventilation can also relieve respiratory distress in patients for whom the work of breathing has become intolerable. In addition, prevention or reversal of atelectasis and reversal of respiratory muscle fatigue can be accomplished by mechanical ventilation.

Even in patients with healthy lungs, mechanical ventilation is often employed when sedation or neuromuscular blockade is necessary (e.g., operative anesthesia). By decreasing systemic or myocardial oxygen consumption, mechanical ventilation may also assist patients who experience compromised myocardial function when the work of breathing becomes excessive. Other objectives of mechanical ventilation include reduction in intracranial pressure by controlled hyperventilation for patients with closed head injury, and stabilization of the chest wall, as in cases of massive flail chest ***(Slutsky, 1993)***.

Generally, mechanical ventilation supplies only symptomatic relief, not actual therapy for respiratory failure or acute lung injury. A notable exception is the state of congestive heart failure, during which mechanical ventilation may augment cardiac output and therefore be therapeutic. Iatrogenic lung injury may be inflicted during mechanical ventilation, and preventive measures should be taken ***(Chamberlain, 2003)***.

# MODES OF MECHANICAL VENTILATION

Studies have demonstrated that suppression of spontaneous breathing and complete dependence on controlled mechanical ventilation lead to rapid respiratory muscle atrophy. Therefore different modes of mechanical ventilation that allow spontaneous breathing, or patient-triggered modes, are favored when feasible ***(Thille, 2006).***

## *Assist/Control Mode Ventilation (AC)*

Assist-control is a commonly used mode of mechanical ventilation in medical intensive care units. A key concept in the AC mode is that the tidal volume (VT) of each delivered breath is the same, regardless of whether it was triggered by the patient or the ventilator. At the start of a cycle, the ventilator senses a patient's attempt at inhalation by detecting negative airway pressure or inspiratory flow. The pressure or flow threshold needed to trigger a breath is generally set by the respiratory therapist and is termed the trigger sensitivity ***(Sassoon et al, 2004)***.

To trigger an assisted breath, the patient must lower the airway pressure by a preset amount, called the trigger sensitivity. **Figure 3** demonstrates examples of pressure and volume waveforms during volume- cycled ACV.

## *Intermittent Mandatory Ventilation (IMV)*

Intermittent mandatory ventilation (IMV) is a type of ventilatory support in which mandatory positive pressure breaths are delivered at preset time intervals. Between these breaths, the patient may breathe spontaneously from a separate circuit containing continuously flowing gas. IMV is associated with patient–ventilator asynchrony because the mandatory breaths are not delivered in concert with the patient's inspiratory effort. A mechanical breath could therefore be delivered during a spontaneous inspiration, leading to lung over distention. IMV has largely been abandoned in favor SIMV ***(Thille, 2006)***.

## *Synchronized Intermittent Mandatory Ventilation (SIMV)*

In SIMV, the patient receives a mandatory number of positive pressure breaths per minute, each of which is synchronized to patient effort. The ventilator can detect the initiation of a spontaneous breath by a patient and does not deliver a machine breath during a spontaneous breath. Between mechanical breaths, however, the patient may breathe an indefinite number of times from a reservoir (**Figure 4).** Spontaneous breaths produce no

response from the ventilator ***(Seymour, 2007)***. **Figure 5** shows typical pressure and volume waveforms during volume-targeted SIMV.

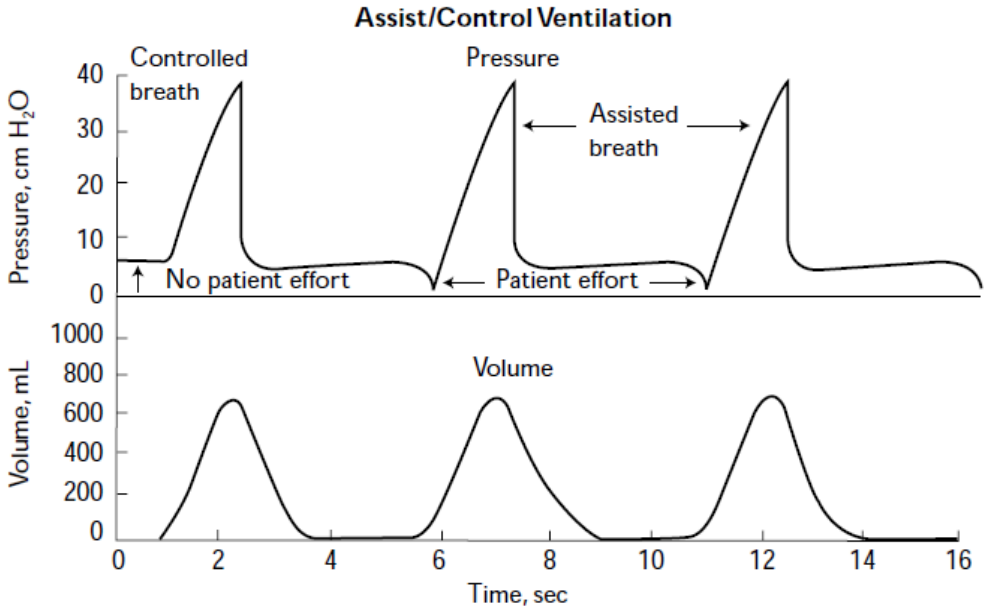



**Figure 3**: pressure and volume waveforms during assist/control ventilation. Note that the fist breath is provided by the ventilator without any patient trigger (controlled breath) and the second and third breaths are triggered by the patient (assisted breaths).H2O=water ***(Seymour, 2007)***

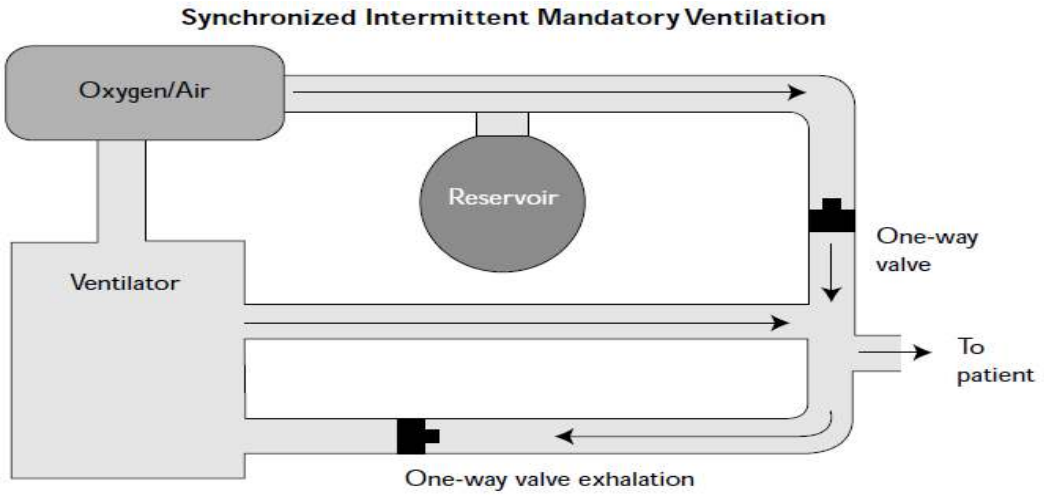



**Figure 4**: A schematic representation of the circuit employed in synchronized intermittent mandatory ventilation ***(Wang et al, 2002)***

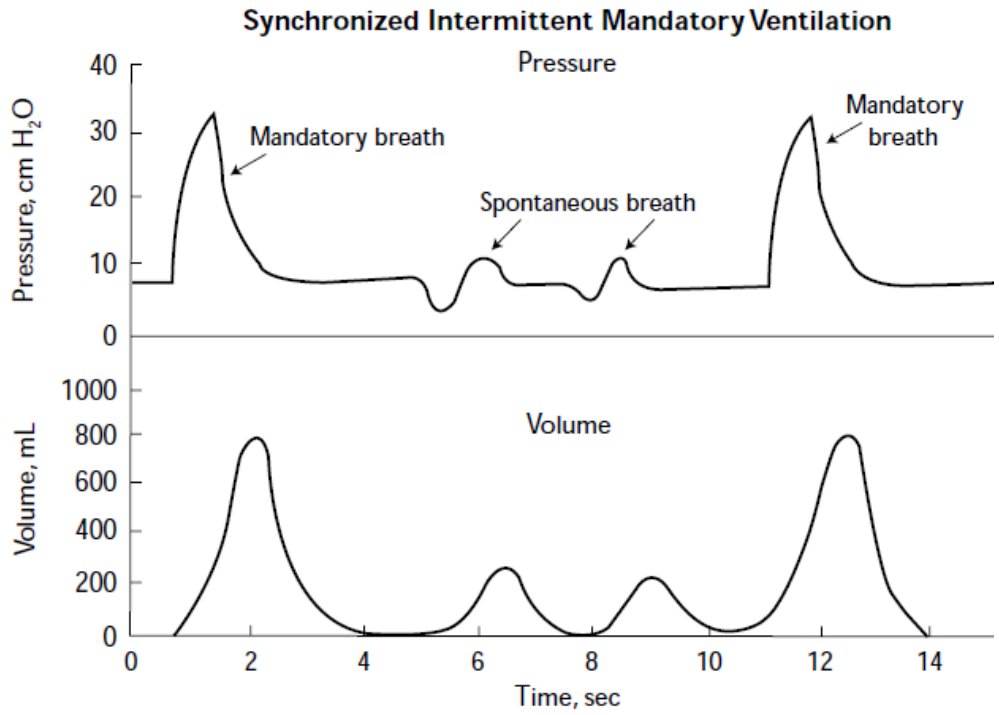



**Figure 5**: pressure and volume waveforms during synchronized intermittent mandatory ventilation. Note that the first and last breaths are mandatory and the second and third are spontaneous.H2O=water ***(Wang et al, 2002)***

## *Pressure-Support Ventilation*

Pressure-support ventilation (PSV) is a relatively new mode of mechanical ventilation. During PSV, each time the patient initiates a spontaneous breath, the negative pressure or flow in the inspiratory circuit opens a valve. The ventilator then delivers a flow of gas sufficient to maintain a constant inflation pressure. Once the patient's inspiratory flow rate falls below a preset threshold level, the flow of gas terminates. With this mode of ventilation, the patient controls respiratory rate and inspiratory time and flow.

Tidal volume and minute ventilation are determined partly by the patient and partly by the ventilator. The tidal volume the patient actually receives depends on the set level of pressure support, patient effort, and the pulmonary mechanics. Pressure support may also be applied to a patient's spontaneous breathing during SIMV. Figure 6 shows pressure and volume waveforms during PSV ***(Chiumello, 2007)***.

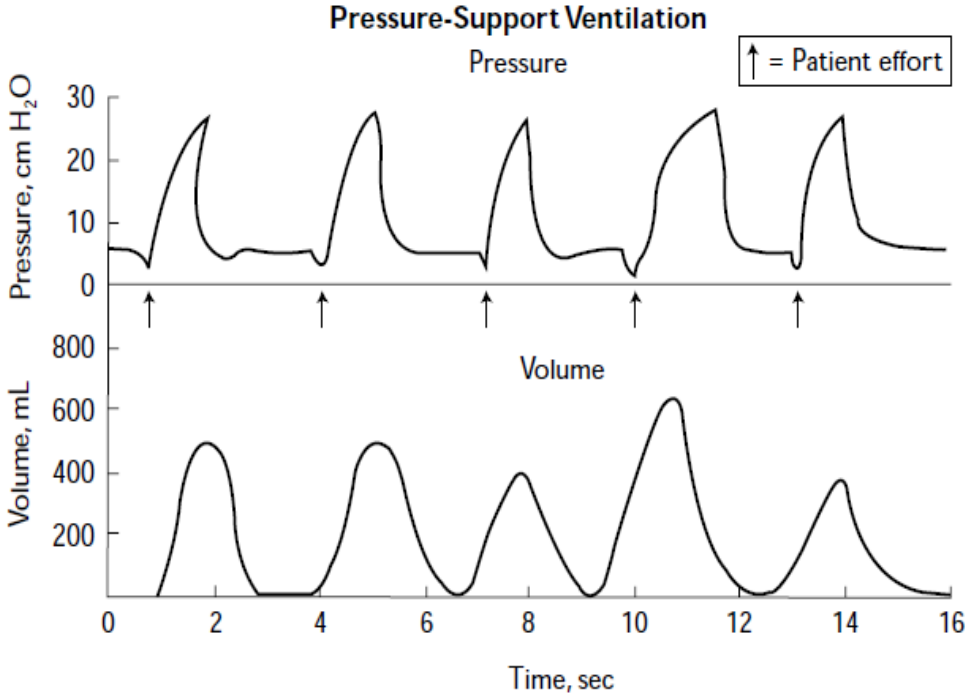



**Figure 6**: pressure and volume waveforms during pressure-support ventilation. Note that each breath is triggered by patient effort. The breaths vary in tidal volume and inspiratory time. H2O=water ***(Tobin, 1994)***

## *SALVAGE MODES OF MECHANICAL VENTILATION*

At times in the course of acute respiratory failure, adequate oxygenation with a nontoxic $Fio_2$ or adequate ventilation cannot be achieved with the modalities of ventilatory support previously described. In these cases, "salvage" modes of mechanical ventilation may be employed.

### *Inverse Ratio Ventilation (IRV)*

Inverse ration ventilation (IRV) is a method of mechanical ventilation in which increases in mean airway pressure (MAP) are developed by prolonging the Inspiratory – to – Expiratory Ratio (I:E ratio). Suggested mechanisms of improved arterial oxygenation with IRV are reduced arteriovenous shunting, improved ventilation-perfusion matching and decreased dead space ventilation ***(Chiumello et al., 2002).***

IRV can be accomplished using either a pressure-controlled or volume-controlled mode of ventilation. Pressure controlled IRV (PC-IRV) is preferred by some authors, who believe the risk of barotrauma induced by auto-PEEP is unacceptably high with volume-cycled IRV (VC-IRV) ***(Mercat et al., 2001).***

***Pressure controlled Inverse Ratio Ventilation:***

Mode of positive pressure ventilation used in patients with more severe respiratory disorders (e.g., adult respiratory syndrome) in which pressure is continuously adjusted downward as lung-compliance and gas exchange improve during inspiration ***(Wang et al., 2002).***

***Volume controlled Inverse Ratio Ventilation:***

Some investigators advocate the use of this mode for the following reasons:

- All adult ventilators can deliver this mode.
- Tidal volume and minute ventilation are guaranteed.
- The I:E ratio can be varied in finer gradations than are usually available with PC-IRV.
- Most clinicians are more familiar with the use of volume-cycled ventilators ***(Prella et al., 2002).***

## *High-Frequency Jet Ventilation (HFJV)*

HFJV is a versatile, safe and effective technique with growing indications for elective and emergency use. This article aims to give the reader an understanding of the basic science necessary to practice. HFJV safely, and increase familiarity with the equipment and techniques used. HFJV is characterized by delivery of small tidal volumes (1-3mls/kg) from a high pressure jet at supraphysiological frequencies (1-10Hz) followed by passive expiration. HFJV is indicated when it offers advantages over conventional ventilation. These indications fall into two main categories; to facilitate surgical access and to optimize pulmonary function ***(Biro et al, 2009).***

## *High-Frequency Oscillatory Ventilation (HFOV)*

High frequency oscillatory ventilation (HFOV) is an alternative form of mechanical ventilation that can be delivered on critical care units. Unlike conventional ventilation (CV), HFOV relies on the rapid delivery of tidal volumes that are smaller than dead space. Typical tidal volumes on HFOV are 1-3 ml/kg. HFOV has been successfully used in neonates and pediatrics since 1983. Studies have shown higher survival rates for patients in these groups with acute respiratory distress syndrome (ARDS). However, the evidence base for the use of HFOV in adults is limited. To date, studies have shown HFOV is safe and equivalent to CV in adult patients with ARDS, but no mortality benefit has been demonstrated. Of note, whether HFOV is superior to CV in adult patients with ARDS is the subject of two

current randomized controlled trials, one in the UK and one in Canada. Despite this lack of a documented mortality benefit there is growing use of HFOV in adult critical care units throughout the UK. In the last five years 26 ventilators were sold in the UK, 10 of which were purchased in 2011 alone. This article will review the basic principles of HFOV and provide practical advice on the selection and clinical management of adult patients ***(Yildizads et al., 2009).***

## *Partial Liquid Ventilation (PLV)*

Partial liquid ventilation (PLV) involves filling the lungs with a fluid. This fluid is perfluorocarbon, also called Liquivent or Perflubron. The liquid has some unique properties. It has a very low surface tension, similar to surfactant, a substance that is produced in your lungs and prevents the alveoli from collapsing and sticking together during exhalation. It also has a high density, oxygen readily diffuses through it, and it may have some anti-inflammatory properties. In PLV, the lungs are filled with the liquid; the patient is then ventilated with a conventional ventilator using a protective lung ventilation strategy. This is called partial liquid ventilation. The hope is that the liquid will help the transport of oxygen to parts of the lung that are flooded and filled with debris, help remove this debris and open up more alveoli improving lung function ***(Kacmarek et al., 2006).***

## *Extracorporeal Membrane Oxygenation (ECMO)*

The term extracorporeal membrane oxygenation (ECMO) was initially used to describe long-term extracorporeal support that focused on the function of oxygenation. Subsequently, in some patients, the emphasis shifted to carbon dioxide removal, and the term extracorporeal carbon dioxide removal was coined. Extracorporeal support was later used for postoperative support in patients following cardiac surgery. Other variations of its capabilities have been tested and used over the last few years, making it an important tool in the armamentarium of life and organ support measures for clinicians. With all of these uses for extracorporeal circuitry, a new term, extracorporeal life support (ECLS), has come into vogue to describe this technology.

The differences between ECMO and cardiopulmonary bypass are as follows:

- ECMO is frequently instituted using only cervical cannulation, which can be performed under local anesthesia; standard cardiopulmonary bypass is usually instituted by transthoracic cannulation under general anesthesia.

- Unlike standard cardiopulmonary bypass, which is used for short-term support measured in hours, ECMO is used for longer-term support ranging from 3-10 days.
- The purpose of ECMO is to allow time for intrinsic recovery of the lungs and heart; a standard cardiopulmonary bypass provides support during various types of cardiac surgical procedures ***(Lowry et al, 2013).***

# ALTERNATIVE MODES OF VENTILATION

***Noninvasive Positive Pressure Ventilation*** **(NIPVV):**

Noninvasive ventilation (NIV) refers to the administration of ventilatory support without using an invasive artificial airway (endotracheal tube or tracheostomy tube). The use of noninvasive ventilation has markedly increased over the past two decades, and noninvasive ventilation has now become an integral tool in the management of both acute and chronic respiratory failure, in both the home setting and in the critical care unit. Noninvasive ventilation has been used as a replacement for invasive ventilation, but its flexibility also allows it to be a valuable complement in patient management ***(Corrado et al., 2009).***

## *Negative Pressure Ventilation (NPV)*

Negative pressure ventilators apply intermittent sub-atmospheric pressure around the chest and abdomen. As the pressure surrounding the chest becomes less than atmospheric, the chest expands and the lungs Inflate. When the pressure surrounding the chest returns to atmospheric pressure, exhalation occurs. The effectiveness of negative pressure ventilators depends upon many variables; the type of system used, degree of patient compliance, the integrity of the airtight seals, and the severity of the patient disease state.

Nocturnal or intermittent ventilatory assistance with negative pressure ventilators has been shown to be effective in reversing mild hypercarbia with several disease states. Iron Lung was successfully used to reverse hypercarbia in patients suffering from neuromuscular diseases. Intermittent use of negative pressure ventilators has also been used with patients suffering from chronic obstructive pulmonary disease (COPD) ***(Keenan et al., 2009).***

## *Airway Pressure Release Ventilation (APRV)*

Airway pressure release ventilation (APRV) is a ventilatory mode that reverses the normal process of breathing. The lungs are kept inflated at a preset pressure level to achieve alveolar distention and recruitment. Exhalation occurs only through cyclic release of the constant airway pressure, which is followed by a rapid restoration. APRV maintains an adequate MAP; functional residual capacity (FRC) is also maintained without requiring an elevated peak airway pressure. Although APRV appears to be effective as a ventilatory mode and theoretically should be beneficial as a lung-protective strategy, no advantages over conventional mechanical ventilation have been demonstrated ***(Porhomayon et al., 2010).***

### *Automatic tube compensation (ATC)*

Some of the newer ventilators in use today include ATC (e.g., Evita XL and Evita, Dräger Medical, Lübeck, Germany; Nellcor Puritan Bennett 840, Puritan Bennett, Pleasanton, California). ATC compensates for resistance associated with an endotracheal tube via closed-loop control of continuously calculated tracheal pressure. A remarkable feature of ATC that makes it superior to PSV is that the intratracheal pressure at the carinal end of the endotracheal tube is used to control flow. In contrast to ATC, in PSV, pressure is generally monitored at the Y-piece or close to the expiration valve of the ventilator. In ATC, intratracheal pressure is calculated on the basis of the patient's inspiratory flow, the circuit pressure, and properties of the endotracheal tube, which are set by the operator in advance. Compared with the situation in PSV, in ATC, tracheal pressure (Ptrach) is relatively constantly maintained. In some ventilators, tube resistance is compensated for during expiration as well as inspiration by a reduction in airway pressure during the expiratory phase ***(MacIntyre et al., 2005).***

# VENTILATOR SETTINGS

### *Tidal Volume (TV)*

Traditionally, an initial tidal volume of 10 mL/kg has been recommended and remains a valid formula for patients without acute lung injury. Recently, however, a large clinical trial was stopped early when fewer deaths occurred among patients with acute respiratory distress syndrome (ARDS) who received small (6 mL/kg) rather than large (12 mL/kg) tidal volumes ***(Hickling, 1990).***

Therefore, in the ARDS population, an initial tidal volume of 6 mL/kg is recommended. Research has suggested that, at least in animal models, overdistention of alveoli by large tidal volumes may actually induce lung damage (e.g., increased pulmonary micro vascular permeability, pulmonary edema, and lung rupture **(*Dreyfuss, 1988).***

Excessive lung distention is of major concern, but direct measurement of the actual volume of air that reaches specific alveoli is not possible. However, lung volume is directly related to the pressure that distends the alveoli. This pressure can be approximated by measuring the end-inspiratory plateau pressure. Plateau pressure is measured by occluding the ventilator circuit at the end of inspiration. Largely based on animal data, a plateau pressure of less than 35 cm water ($H_2O$) has been recommended as safe ***(Slutsky, 1993)***.

To achieve this target, tidal volume may need to be decreased to below 5 mL/kg, the physiologic value for spontaneous breathing. Low tidal volumes may lead to decreased ventilation and an elevated arterial partial pressure of carbon dioxide ($PaCO_2$), a ventilatory strategy referred to as permissive hypercapnia ***(Hickling, 1990)***.

When using permissive hypercapnia, clinicians are advised to focus on the pH, rather than the $PaCO_2$. Although some authorities recommend the administration of intra-venous bicarbonate once the pH is below 7.2, this practice remains controversial ***(Hewlett, 1977)***.

## *Respiratory Rate (RR)*

The ideal mechanical respiratory rate depends on the particular mode of ventilation. When using ACV, the respiratory rate should be approximately 4 breaths/min less than the patient's spontaneous respiratory rate, thus assuring adequate ventilation if the patient's spontaneous efforts cease. If SIMV is employed or if the patient has no spontaneous respirations, the rate should be initially set at 10 breaths/min; respiratory rate should be increased if higher minute ventilation is needed (as with respiratory acidosis) and decreased gradually as patient tolerance permits. No respiratory rate is set for PSV ***(Marini, 1989)***.

## *Sensitivity*

To trigger an assisted breath during ACV or a pressure-supported breath during PSV, a patient must lower airway pressure by a preset amount to open the "demand valve" that delivers gas. The required change in airway pressure (i.e., trigger sensitivity) is typically set from –1 to –3 cm $H_2O$. The actual negative pressure a patient must generate, however, can be much higher if the demand valve is poorly responsive. Conversely, if the trigger sensitivity is set too low, the ventilator may deliver breaths too frequently (commonly referred to as auto-cycling) and produce severe respiratory alkalosis. Some ventilators may be triggered by inspiratory flow rather than pressure. This method of triggering appears to require less patient effort ***(Dreyfuss, 1988).***

## *Fraction of Inspired Oxygen (FiO2)*

Inhalation of any gas with a fraction of inspired oxygen ($Fio_2$) higher than the $Fio_2$ of room air can potentially be toxic. However, 0.6 has been established as the threshold of a toxic concentration of $Fio_2$. To minimize the risk of toxicity, the lowest $Fio_2$ that achieves adequate oxygenation should be employed. Acceptable arterial oxygen saturation is usually defined as 90% or greater, which usually corresponds to an arterial partial pressure of oxygen ($PaO_2$) of approximately 60 mm Hg or more***(Amato, 1998)***.

## *Positive End-Expiratory Pressure (PEEP)*

In individuals with healthy lungs, the intrathoracic pressure at the end of exhalation closely approximates atmospheric pressure. Intrapleural pressure, however, is slightly negative. This negative pressure keeps a resting volume of air inside the lung at the end of exhalation, called the FRC. FRC provides a reservoir of gas that maintains a constant $PaO_2$ and $PaCO_2$, a crucial physiologic function.

Patients with respiratory failure who require mechanical ventilation, however, usually have a reduced FRC as a consequence of surfactant loss and alveolar instability, which leads to alveolar collapse. In turn, alveolar collapse can lead to severe and rapid hypoxemia that is reversed by artificially increasing airway pressure at the end of expiration to force the alveoli open and maintain a greater lung volume.

Improvement in oxygenation correlates with the mean airway pressure, which is defined as the airway pressure averaged over time. Therefore, methods that raise the mean airway pressure also improve oxygenation. Positive end-expiratory pressure (PEEP) is the most commonly employed method to elevate mean airway pressure and improve oxygenation (Figure 5).

PEEP and continuous positive airway pressure, which is PEEP applied to spontaneous breaths, may enhance oxygenation and allow reduction in $Fio_2$ with its attendant risk of toxicity. Excessive PEEP, however, may reduce cardiac output and impair systemic oxygen delivery, thus offsetting any improvement in oxygenation. The application of PEEP has traditionally been used to improve oxygenation in patients with respiratory failure caused by various conditions, especially ARDS. Use in patients with acute respiratory distress syndrome. PEEP improves oxygenation in patients with ARDS by decreasing intrapulmonary shunting his decrease is accomplished in several ways, including the recruitment of collapsed alveoli, increase in FRC, and redistribution of lung water from the alveoli into the perivascular interstitial space (***Malo, 1984).***

Patients with ARDS typically have a decreased end- expiratory lung volume, so that tidal breathing takes place on the lower, flat part of the pressure-volume curve. By increasing tidal volume, the application of PEEP moves tidal breathing to a part of the curve with higher compliance and decreases the work of breathing in one group of patients with ARDS, survival rates at 28 days improved when PEEP was applied to keep the alveoli open as a part of a protective ventilatory strategy ***(Amato, 1998)***.

## *Auto–Positive End-Expiratory Pressure (auto PEEP)*

Some patients with asthma or COPD may develop air trapping caused by flow limitation and inadequate emptying of the lungs during exhalation. This higher lung volume at the end of exhalation leads to an increased end-expiratory pressure called auto-PEEP. Auto-PEEP (Figure 7) is measured by occluding the expiratory port of the ventilator during the last 0.5 seconds of exhalation. The auto-PEEP measurement is extremely sensitive to timing. Several trials are usually needed to achieve proper timing with respect to the ventilatory cycle and patient effort.

Modern ventilators measure auto-PEEP automatically. Older ventilator models cannot sense auto-PEEP, and the patient must decrease airway pressure by an amount equal to auto PEEP plus the trigger sensitivity in order to trigger the ventilator. Auto-PEEP may therefore cause difficulty triggering the ventilator during spontaneous breaths, which may lead to an excessive work of breathing and respiratory muscle fatigue in some patients. The normal triggering effort during spontaneous breaths can be returned by adding extrinsic PEEP to a level just below the auto-PEEP level ***(Smith, 1988)***.

Application of extrinsic PEEP is not helpful in patients who are not breathing spontaneously. Not all patients with detectable auto-PEEP experience air trapping and hyperinflation. Auto-PEEP may occur without air trapping during forcible exhalation. Auto-PEEP and air trapping may also occur without flow limitation if the patient's ventilatory requirements are very high and the time for full exhalation is not sufficient. High resistance to exhalation, as demonstrated in patients with small endotracheal tubes, may also cause auto -PEEP without air trapping. In such cases, the addition of extrinsic PEEP does not decrease the work of breathing and may be detrimental ***(Marini, 1989)***. If extrinsic PEEP is applied to patients with true airflow limitation, airway pressures must be monitored. If, after the addition of extrinsic PEEP, the peak and plateau airway pressures do not rise during a passive machine- delivered breath, flow limitation is present and the extrinsic PEEP may be potentially beneficial. In other patients, extrinsic PEEP has the potential to worsen hyperinflation and cause volume-related trauma and hemodynamic compromise ***(Tuxen, 1989)***.

## *Inspiratory Flow Rate (IFR)*

During ACV and SIMV, the inspiratory flow rate is often set at 60 L/min on a mechanical ventilator. Patients with COPD, however, may attain better gas exchange with flow rates as higher flow rates allow the delivery of a tidal volume in a shorter period of time and leave a longer period of time for exhalation, which favors more complete lung emptying and less gas trapping. With higher flow rates, however, peak inspiratory pressure also increases and may exceed the ventilator safety limits, which reduces delivered tidal volume. If the inspiratory flow rate is too slow to meet the ventilatory requirements, the patient generates highly negative intrapleural pressure by inhaling against a closed inspiratory valve. The negative intrapleural pressure may lead to muscle fatigue and even pulmonary edema ***(Marini, 1985)***.

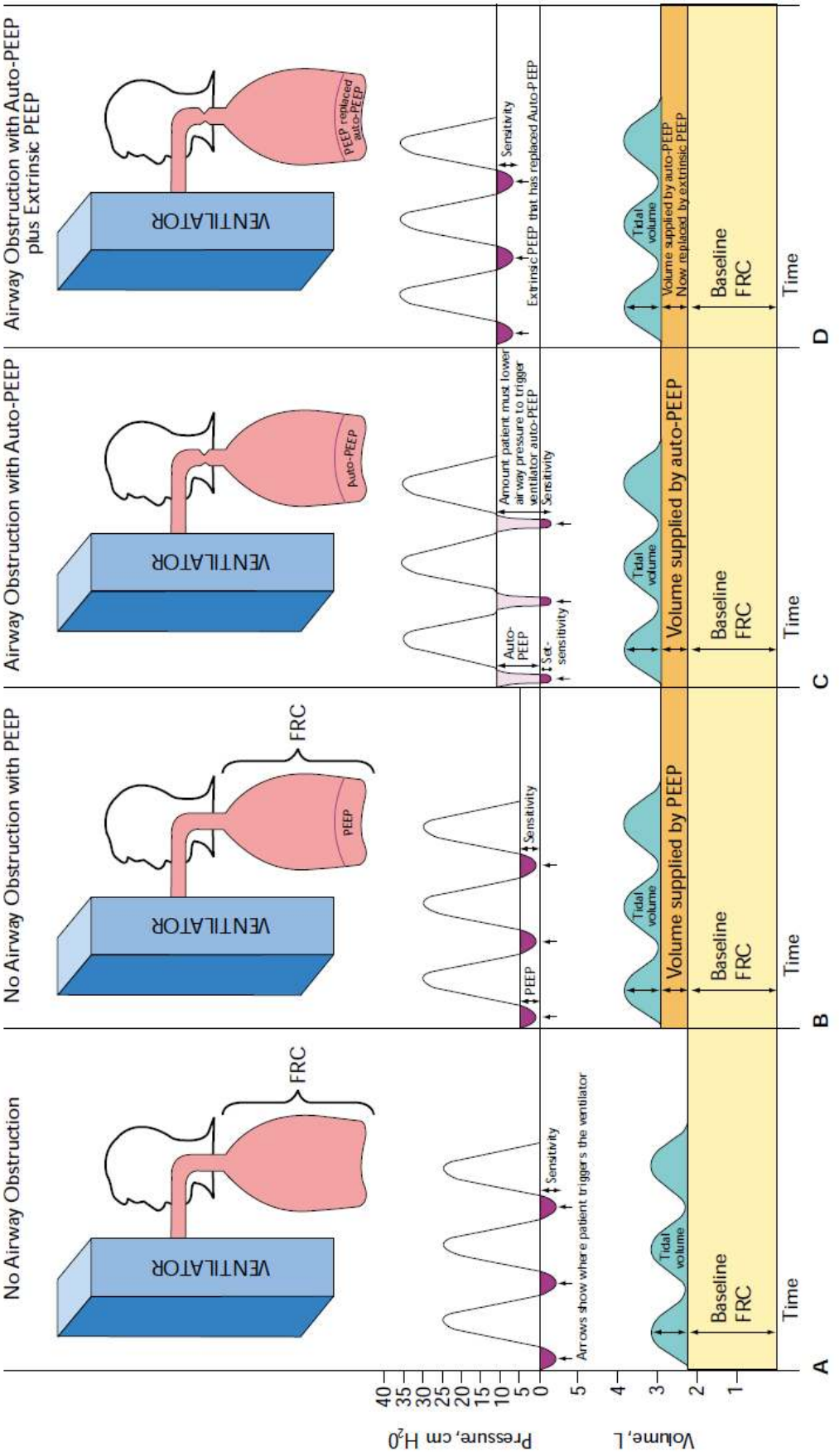



**Figure 7**: A schematic illustration of positive end-expiratory pressure ***(Tobin, 1994)***

A) During mechanical ventilation with extrinsic PEEP. Patients without airway

obstruction exhale to a new FRC that equals the baseline FRC plus the volume added by PEEP. To trigger the ventilator, patients must lower airway pressure below PEEP by a level equal to sensitivity.

B) During mechanical ventilation without extrinsic PEEP, patients with airway obstruction may not be able to exhale to FRC leading to air trapping. The trapped air creates auto-PEEP. Because the ventilator cannot sense the auto-PEEP, spontaneously breathing patients must lower the air way pressure by a level equal to sensitivity plus auto-PEEP to trigger the ventilator which may take considerable effort.

C) During mechanical ventilation without extrinsic PEEP, patients with airway obstruction may not be able to exhale to FRC leading to air trapping. The trapped air creates auto-PEEP. Because the ventilator cannot sense the auto-PEEP, spontaneously breathing patients must lower the airway pressure by a level equal to sensitivity plus auto-PEEP to trigger the ventilator which may take considerable effort.

D) In the presence of auto-PEEP, application of extrinsic PEEP approximately equal to auto-PEEP may replace auto-PEEP without increasing lung volume. To trigger the ventilator, patients must lower airway pressure only to a level equal to sensitivity.

# PATHOPHYSIOLOGICAL CONSEQUENCES OF MECHANICAL VENTILATION

## *Achieving a ventilation/perfusion balance*

Each lung unit consists of approximately 150 million alveoli, which are ventilated according to their position on the pressure-volume curve (Figure 8). Transpulmonary pressure is defined as airway pressure minus pleural pressure. The dependent regions of the lungs are subject to a lower transpulmonary pressure. This region is therefore positioned on the lower steeper part of the pressure–volume curve. When a negative intrathoracic pressure and generated during normal spontaneous ventilation, the transpulmonary pressure is increased to an equal extent all over the lungs. Thus the changes in transpulmonary pressure is greatest and the dependent areas making this part most compliant and hence causing it to receive the most ventilation ***(Walker, 2000)***.

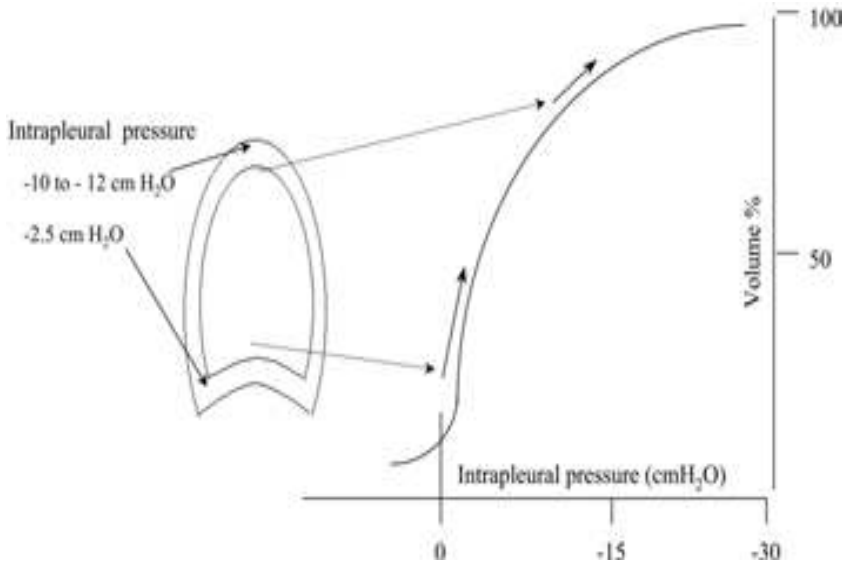



**Figure 8**: Intrapleural pressure and ventilation in upright lung ***(Kihara, 2003)***

Similarly, blood flow is also unevenly distributed, mainly influenced by gravitational forces. The lungs can be divided into arbitrary zones in order to understand the effect of gravity and influence of alveolar pressure on the blood flow (Figure 9). The upper region of the lungs (zone I) may receive very little blood as alveolar pressure may exceed arterial and venous pressures. Zone II lung arterial pressure is greater than the alveolar pressure which in turn is greater than the venous pressure. Blood flow is thus dependent on alveolar pressure ***(Kihara, 2003)***.

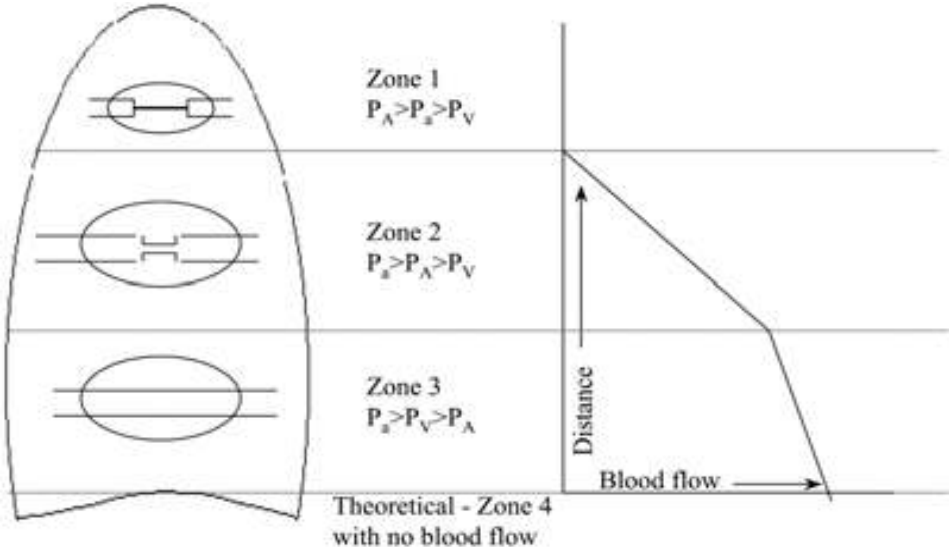



**Figure 9:** Distribution of pulmonary blood flow in the zone model of the lung ***(Kihara, 2003)***

In zone III, both arterial and venous pressures exceed alveolar pressure and it is in this region that the best alveolar blood flow is seen. In zone IV; the interstitial pressure may physically compress arterioles and reduce the blood flow. In a state of low cardiac output, blood flow can almost cease in zone I. This is due to a decrease in pulmonary artery pressure whilst the alveolar pressure remains unaffected, effectively increasing dead space ***(Varelas, 2002)***.

The change from normal breathing to mechanical ventilation can cause profound alterations in respiratory physiology. Ventilated patients are typically nursed in a supine position, which results in a decrease in total lung volume and functional residual capacity. Closing capacity encroaches on tidal ventilation meaning airways start to close during normal ventilation. This effect causes an increase in the amount of physiological shunting and potentially a worsening hypoxemia.

Mechanical ventilation can also adversely affect ventilation/perfusion matching because ventilation occurs predominantly in nondependent areas whereas blood flow is concentrated in dependent regions ***(Berrouschot, 2000)***.

Cardiovascular changes are extremely common when a patient is initially placed on a ventilator and these are predominantly caused by an increase in intra-thoracic pressure leading to a reduction in venous return to the heart. This reduction in 'preload' can cause a dramatic reduction in cardiac output leading to profound hypotension, especially in a patient who is hypovolemic before ventilation is

instituted. These changes may cause a paradoxical increase in cardiac output in patients with heart failure due to a beneficial instant reduction in preload.

Mechanical ventilation should not be taken lightly as elevated intra-thoracic pressure affects regional hemodynamics and may lead to dysfunction of further organs. These are due to a combination of factors:

1- Up to 30% reduction in cardiac output and systemic arterial pressure.

2-Increase in intra-thoracic pressure leading to impairment of venous return and elevation of venous pressure.

3-Shearing injury may leads to modification of local vasoactive status ***(Montravers, 2002)***.

# MONITORING DURING MECHANICAL VENTILATION

Several advances in monitoring gas exchange, neuromuscular capacity, respiratory mechanics, and patient effort during mechanical ventilation have occurred in recent years. Monitoring these parameters is helpful in minimizing ventilator-induced complications, optimizing patient-ventilator interaction and determining a patient's readiness for the discontinuation of mechanical ventilation ***(Jubran, 1998).***

**Gas Exchange**

With the proliferation of pulse oximeters in different locations of the hospital throughout the 1980s, several investigators demonstrated that episodic hypoxemia is much more common than previously suspected, with an incidence ranging from 20 to 40%. In patients admitted to a general medical service, it was found that patients who experienced hypoxemia (O2 saturation, 90% for ≥ 5 min) during the first 24 hrs. of hospitalization had a mortality rate more than three times higher than patients who did not experience desaturation. Whether or not the early detection and treatment of episodic hypoxemia can affect patient outcome remains to be answered ***(Stewart, 1998).***

**Pulse Oximetry**

Pulse Oximetry is based on two physical principles:

(1) The presence of a pulsatile signal generated by arterial blood, and

(2) The fact that ox hemoglobin (O2Hb) and reduced hemoglobin (Hb) have different absorption spectra ***(Ortiz, 1999)***.

Currently available oximeters use two light-emitting diodes that emit light at the 660 nm (red) and the 940 nm (infrared) wavelengths. These two wavelengths are used because O2Hb and Hb have different absorption spectra at these particular wavelengths. In the red region, O2Hb absorbs less light than Hb, while the reverse occurs in the infrared region. The ratio of absorbencies at these two wavelengths is calibrated empirically against direct measurements of arterial oxygen saturation (Sao2) in volunteers, and the resulting calibration algorithm is stored in a digital microprocessor within the pulse oximeters ***(Stewart, 1998)***.

## *Capnography*

The end-tidal Pco2 concentration (PETco2) is the value of exhaled gas taken at the plateau of the CO2 waveform. Consequently, PETco2 can be employed as a continuous, indirect measure of Paco2 ***(Malatesha, 2007)***.

## *Respiratory Neuromuscular Function*

### Airway Occlusion Pressure

Measuring mouth occlusion pressure at 0.1second after onset of inspiratory effort against an occluded airway (P0.1) provides a measure of respiratory drive. In ventilator-dependent patients, P0.1 has been shown to correlate significantly with work of breathing (WOB) during pressure-support ventilation (PSV).Several studies have indicated that an elevated P0.1 predicted weaning failure, but the threshold separating success from failure differed among the studies.

### Breathing Pattern

Minute ventilation should be partitioned into tidal volume (TV) and respiratory frequency (f). In healthy subjects, f is approximately 17 breaths/min and TV is approximately 400 ml. An elevated frequency is often the earliest sign of impending respiratory distress, and the degree of elevation is proportional to the severity of the underlying lung disease. Rapid shallow breathing is a common finding in patients who fail a trial of weaning from mechanical ventilation and this can be quantitated in terms of the f/VT ratio; a value. 100 breaths/ min/L suggests that a trial of weaning is unlikely to be successful. Rapid shallow breathing has been considered a useful strategy to avoid fatigue during a failed weaning trial. However, rapid shallow breathing develops immediately following the discontinuation of mechanical ventilation and does not progress with time-a response that is difficult to attribute to fatigue. Moreover, data in patients failing a weaning trial indicate a poor correlation between f/VT and the tension-time index, a crude index of impending respiratory muscle fatigue. To serve as a compensatory strategy to avoid fatigue, f/VT should have a negative correlation with tension-time index, whereas r was found to be 0.08 ***(Keenan, 2003)***.

**Maximal Inspiratory Airway Pressure**

Global inspiratory muscle strength is assessed by measuring maximal inspiratory pressure while the patient makes a maximum inspiratory effort against an occluded airway, preceded by complete exhalation to residual volume. To obtain more reproducible recordings, a two-step modification was introduced consisting of a one-way valve to ensure that inspiration begins at a low lung volume and maintaining the period of occlusion for.

Maximal inspiratory pressure is one of the standard measurements employed to determine a need for the continuation of mechanical ventilation. Values that are more negative than 230 cm H2O are thought to predict weaning success, while values that are less negative than 220 cm H2O are predictive of weaning failure. However, these criteria are frequently falsely positive and falsely negative ***(Meade, 2008)***.

## *Respiratory Mechanics*

Measurements of respiratory mechanics in a relaxed ventilator-dependent patient can be obtained using the technique of rapid airway occlusion during constant flow inflation. Rapid airway occlusion at the end of a passive inflation produces an immediate drop in both airway pressure (Paw) and trans pulmonary pressure (Pl) from a peak value (Ppeak) to a lower initial value (Pinit) followed by a gradual decrease until a plateau (Pplat) is achieved after. Pinit is measured by back extrapolation of the slope of the latter part of the pressure tracing to the time of airway occlusion. Pplat on the Paw, Pl, and pleural pressure (Pes) tracings represents the static end-inspiratory recoil pressure of the total respiratory system, lung, and chest wall, respectively.

**Elastance**

The end-inspiratory airway occlusion method is clinically used to measure the static compliance of the respiratory system or its reciprocal, elastance of the respiratory system (Est,rs), according to the following equation:

*Est,rs =(Pplat x PEEPi)/VT*

Where Pplat is plateau pressure obtained after occluding the airway, PEEPis intrinsic positive end expiratory pressure (PEEP), and VT is tidal volume. Using an esophageal balloon catheter, Est,rs can be partitioned into its lung and chest wall components by dividing [Pplat 2 PEEPi] by VT on the Pl and Pes tracings,

respectively. In mechanically ventilated patients with acute respiratory failure secondary to COPD or pulmonary edema, Est,rs is higher than in normal subjects. Static lung elastance is higher in patients with pulmonary edema than in patients with COPD, whereas static chest wall elastance was similar in both patient groups ***(Brower, 2003)***.

**Dynamic Compliance**

An index that commonly is referred to as effective dynamic compliance, or the dynamic characteristic, can be derived by dividing the ventilator delivered VT by [peak Paw x PEEP]. This index is not a measure of true thoracic compliance because peakPaw includes all of the resistive and elastic pressure losses of the respiratory system and endotracheal tube. Alternatively, dynamic elastance of the respiratory system (Edyn,rs) can be obtained by dividing the difference in Paw at points of zero flow by the delivered VT. Accordingly, Edyn,rs can be computed according to the formula: Edyn,rs = Pinit x PEEPi/Vt.

Edyn, rs can be partitioned into its lung (Edyn,L) and chest wall components by dividing [Pinit x PEEPi] on Pl and Pes tracings, respectively. In a recent study, Edyn, rs was found to be similar in patients with COPD who went on to fail a trial of spontaneous breathing and in a control group who tolerated the trial and were extubated. In both groups, Edyn,rs was predominantly influenced by Edyn,L because the values of chest wall dynamic elastance were normal. Edyn,L was significantly higher in the failure group than in the success group, but the individual values showed a considerable overlap among the patients in the two groups, thus limiting its usefulness in signaling a patient's ability to sustain spontaneous ventilation ***(Hatlestad, 2003)***.

**Pressure-Volume Curves**

A pressure-volume curve of the respiratory system can be constructed in a paralyzed patient by measuring the airway pressure as the lungs are progressively inflated with a 1.5 to 2 L syringe. A lower inflection point and an upper inflection point may be seen on the pressure-volume curve. The lower inflection point is thought to reflect the point at which small airways or alveoli reopen, corresponding to closing volume. In patients with acute lung injury, some investigators have recommended that PEEP should be set at a pressure slightly above the lower inflection point.

**Resistance**

Airway resistance can be measured in ventilatordependent patients by using the technique of rapid airway occlusion during constant flow inflation. The maximum resistance (Rmax) and minimum resistance (Rmin) of the total respiratory system, lungor chest wall can be computed by dividing [Ppeak x Pplat] and [Ppeak x Pinit] from the Paw, Pl, and Pes tracings, respectively, by the flow immediately preceding the occlusion. The additional resistance (DR) of the respiratory system, lung, or chest wall can be calculated as Rmax x Rmin for the respiratory system, lung, or chest wall, respectively. Rmin is considered to reflect ohmic airway resistance while DR reflects both the viscoelastic properties (stress relaxation) and time-constant in homogeneities within the respiratory tissues (pendelluft). Measurements of airway resistance are helpful in assessing the response of patients to bronchodilator therapy ***(Marelich, 2000)***.

**Intrinsic PEEP**

The static recoil pressure of the respiratory system at end expiration may be elevated in patients receiving mechanical ventilation. This positive recoil pressure, or intrinsic PEEP (static PEEPi), can be quantified in relaxed patients by using an end expiratory hold maneuver on a mechanical ventilator immediately before the onset of the next breath. PEEPi poses a significant inspiratory threshold load that has to be fully counterbalanced by increasing inspiratory muscle effort in order to generate a negative pressure in the central airway and trigger the ventilator. Thus, PEEPi adds to the triggering pressure such that the total inspiratory effort needed to trigger the ventilator is the set trigger sensitivity plus the level of PEEPi. This is one of the factors that may account for the not infrequent observation of a patient who is unable to trigger a ventilator despite obvious respiratory effort.

In a spontaneously breathing patient, an esophageal balloon catheter system can be used to measure PEEPi during un-occluded breathing (dynamic PEEPi).This is achieved by calculating the negative deflection in esophageal pressure from the start of inspiratory effort to the onset of inspiratory flow. To obtain valid measurements, both the inspiratory and expiratory muscles need to be relaxed at end expiration. Two methods have been proposed to distinguish between the contribution of elastic recoil and expiratory muscle activity to PEEPi, with the latter being estimated from

measurement of either the increase in gastric pressure (Pga) over the course of expiration or the decrease in Pga at the onset of the next expiration ***(Perez, 2000)***.

**Work of Breathing (WOB)**

The mechanical WOB can be calculated by measuring the generation of intrathoracic pressure due to contraction of the respiratory muscles (or a ventilator substituting for them) and the displacement of gas volume. Coussa et al found that inspiratory work was approximately twofold greater in patients with COPD receiving controlled mechanical ventilation than in healthy control subjects, and the difference between the two groups was almost completely explained by PEEPi. Likewise, in patients with COPD receiving PSV, PEEPi accounted for 63% of the total amount of patient effort. A number of investigators have examined the usefulness of respiratory work measurements in predicting the outcome of a trial of weaning from mechanical ventilation. These studies show that patients can tolerate only a very small fraction of the maximum possible workload. Furthermore, WOB appeared to be higher in ventilator-dependent patients compared with ventilator-independent patients. Unfortunately, the predictive value of respiratory work as an index of weaning outcome remains to be determined (***Martin, 1991)***.

**Pressure-Time Product (PTP)**

A significant limitation of measurements of respiratory work is that they underestimate energy expenditure during isometric contractions. To overcome this problem, many investigators have measured pressure-time product (PTP) during mechanical ventilation. This is calculated as the time integral of the difference between esophageal pressure (Pes) measured during assisted breathing and the recoil pressure of the chest wall measured during passive ventilation with VT and flow settings that are identical to the assisted breaths. While this can be achieved satisfactorily during assist-control ventilation and intermittent mandatory ventilation, a problem arises during PSV because lung volume and inspiratory flow vary from breath to breath in this mode.

To overcome this problem, a modified approach in the calculation of PTP has been described First, an estimated recoil pressure of the chest wall is quantitated on a breath-by-breath basis by multiplying chest wall elastance (measured during passive ventilation) by lung volume. Then PTP is calculated as the time integral of the difference between the Pes tracing and the recoil pressure of the chest wall.

An element of uncertainty exists with the measurement of PTP, however, because the rapid decrease in Pes before the onset of inspiratory flow may result from inspiratory muscle activity needed to overcome the threshold imposed by dynamic hyperinflation and/or cessation of expiratory muscle activity. To deal with this issue, an upper- and lower-bound inspiratory PTP can be calculated that includes the entire possible range of muscular activity ***(Zimmerman, 2001)***.

To calculate upper-bound inspiratory PTP, the estimated elastic recoil pressure of the chest wall was set equal to Pes at the onset of the rapid decrease in Pes. To calculate lower-bound inspiratory PTP, the estimated elastic recoil pressure of the chest wall is set equal to Pes at the onset of inspiratory flow. In patients with COPD, a marked and progressive decrease in upper-bound PTP was observed during graded levels of PSV, but the response among patients was quite variable, with a coefficient of variation up to 96%.Evidence of expiratory effort, quantitated by an expiratory PTP, was seen in many patients, and this increased as PSV was increased.

Moreover, several patients displayed expiratory muscle activation during late inflation, indicating that the patient was fighting the ventilator. This was more common in patients who had elevated time constants and who required more time for inspiratory flow to fall to the threshold value required for termination of inspiratory assistance by the ventilator (25% of peak inspiratory flow) ***(Strand, 2008)***.

# COMPLICATIONS OF MECHANICAL VENTILATION

Mechanical ventilatory support is by no means risk free, and one of the principal goals of mechanical ventilation is avoidance of complications. Close monitoring is necessary to identify patients at risk for or in the early phases of iatrogenic complications. Patients should also be continuously tested to detect when they are able to discontinue mechanical ventilatory support, because the incidence of most problems is correlated to the duration of mechanical ventilation. Complications can be related to the endotracheal tube (i.e., laryngeal injury, tracheal stenosis, tracheomalacia, or sinusitis).

Barotrauma, such as pneumo-thorax, subcutaneous emphysema, or pneumomediastinum, occurs in approximately 10% to 20% of mechanically ventilated patients. Barotrauma is probably related to asymmetric alveolar overdistention in addition to high airway pressures. Because mechanically ventilated patients are usually supine, the classic apical or apicolateral presentation of pneumothorax occurs less frequently than in non-ventilated patients and the diagnosis is often missed.

Positive pressure ventilation may cause diminished venous return and, in turn, decrease cardiac output in patients with normal myocardial function. Patients with impaired myocardial contractility, however, can experience increased cardiac output because left ventricular afterload is decreased when intra-thoracic pressure increases and, conversely, transmural aortic pressure decreases.

Ventilator-associated pneumonia is an ominous development that carries a crude mortality rate of approximately 30%.Non-pharmacologic strategies to prevent ventilator-associated pneumonia include ***(Kress, 2002)***:

- Proper hand washing by caregivers
- Semi-recumbent positioning of the patient to prevent aspiration
- Adequate nutritional support
- Avoidance of gastric distention
- Early removal of endotracheal and nasogastric tubes
- Continuous subglottic suctioning

Although maintenance of adequate pressure in the endotracheal tube cuff is also recommended, cuff pressure above 25 mm Hg may affect perfusion of the tracheal mucosa and result in injury ***(Tobin, 1994).***

The use of sucralfate in place of histamine 2-receptor antagonists or antacids for stress-ulcer prophylaxis is associated with lower rates of ventilator-associated pneumonia. Other pharmacologic interventions that have been advocated for prevention of ventilator-associated pneumonia include: avoidance of unnecessary antibiotics, use of chlorhexidine oral rinse, and use of prophylactic antibiotics and granulocyte colony-stimulating factor in ventilated patients with neutropenic fever ***(Jubran, 1993).***

# SEDATION OF MECHANICALLY VENTILATED PATIENTS

Sedatives and analgesics are the most commonly administered medication in both surgical and medical ICU accounting for 10-15% of the total drug costs. Daily interruption of sedative infusions and complications of critical illness in mechanically ventilated patients ***(Esteban, 1999)***.

In an attempt to improve sedation and analgesia in our ICU patients, thereby improving patient outcome and costs the following guidelines have been created. These guidelines are based on recommendations developed by the Society of Critical Care Medicine (January 2002) combined with data on the pharmacodynamics and pharmacokinetics of the drugs in the critically ill ***(Esteban, 1995)***.

## *Ramsay Scale*

Sedation level Description

1) Anxious and agitated
2) Cooperative, tranquil, oriented
3) Responds only to verbal commands
4) Asleep with brisk response to light stimulation
5) Asleep without response to light stimulation
6) Non responsive ***(Jubran and Tobin, 1997).***

## *Richmond Agitation Sedation Scale (RASS)*

Target RASS Description.

+ 4 Combative, violent, danger to staff
+ 3 Pulls or removes tubes or catheters; aggressive
+ 2 Frequent non purposeful movement, fights ventilator
+ 1 Anxious, apprehensive, but not aggressive
0 Alert and calm
- 1 Awakens to voice (eye opening/contact) >10 sec
- 2 Light sedation, briefly awakens to voice (eye opening/contact) <10 sec
- 3 Moderate sedation, movement or eye opening. No eye contact
- 4 Deep sedation, no response to voice, but movement or eye opening to physical stimulation
- 5 Unarousable, no response to voice or physical stimulation ***(Ely et al, 1999).***

# WEANING FROM MECHANICAL VENTILATION

The term weaning is commonly used to describe the process of removing a patient from mechanical ventilation and restoring spontaneous breathing. In some cases (e.g., patients recovering from drug overdose or operative anesthesia), ventilatory support may simply be removed when clinical observations suggest that the patient is capable of resuming spontaneous breathing. In other cases, especially in patients recovering from ARDS or severe COPD, weaning can be a labor-intensive and time-consuming endeavor.

**Anticipation of Weaning**

Discontinuation of mechanical ventilation is generally not considered until the patient has achieved hemodynamic and cardiopulmonary stability. Additionally, many authors recommend that the patient should at least have a $PaO_2$ greater than or equal to 60 mm Hg while breathing, an $Fio_2$ less than or equal to 40%, and a PEEP less than or equal to 5 cm $H_2O$.

Ideally, the patient should be awake and alert, have adequate and stable hemoglobin and renal function, and be free of the need for vasoactive or sedative agents. The patient should also be afebrile or febrile and hemodynamically stable, have an intact respiratory drive, and have the ability to protect the upper airway and adequately clear secretions.

**Standardized Weaning Strategies**

Current recommendations about managing the weaning process emphasize the need for early testing of the patient's ability to breathe spontaneously. Daily determination of simple criteria has been suggested to assess readiness for weaning **(*Ely et al, 1999*).** These criteria can be evaluated by physicians, nurses, specialized physical therapists, or respiratory therapists.

**The criteria for weaning a patient from mechanical ventilation:**

- Adequate oxygenation PaO2 ≥ 60 mmHg on FiO2 ≤ 0.4 (PaO2/FiO2 = 150-300) with PEEP ≤ 5 cm H2O.
- Hemodynamic stability.
- No myocardial ischemia or clinically significant hypotension.
- Temperature < 38°C.

- No significant acid-base disturbance (i.e., absence of respiratory acidosis).
- Hemoglobin ≥ 8-10 g/dL.
- Adequate mental status (Patient awake or easily aroused).
- Stable metabolic status (e.g., acceptable electrolytes).
- Able to initiate an inspiratory effort.

**Prediction of Weaning Success**

Successful discontinuation of mechanical ventilation depends on large part on the ability of the respiratory muscles to tolerate a workload that is usually elevated as the lungs recover ***(Unroe, 2010)***.

Jubran and Tobin reported that a detailed evaluation of respiratory mechanics in patients receiving controlled ventilation was unable to discriminate between patients who failed a weaning trial and those who were weaned successfully **(*Jubran and Tobin, 1997*)**. Therefore, a spontaneous breathing trial should be performed routinely regardless of the level of ventilator support. Patients who meet the criteria for starting weaning should undergo trial of spontaneous breathing. Two main methods are used ***(Esteban, 1997)***.

1) The T-tube test involves disconnecting the ventilator and supplying oxygen and moisture via a T-tube connected to the endotracheal tube, with the amount of supplemental oxygen set to achieve ≥ 92% arterial oxygen saturation.
2) Spontaneous breathing can be assessed as the patient receives a low level of inspiratory pressure support for instance, of 7 to 10 cm H2O according to whether a moisturizing filter (artificial nose) is used. Positive end-expiratory pressure is probably inadvisable, although this point is not universally agreed on.

**Criteria to determine success of a trial of spontaneous breathing:**

- *Objective criteria*
  - SaO2 > 90% or PaO2 > 60 mmHg on FiO2 < 0.4-0.5
  - Increase in PaCO2 < 10 mmHg or decrease in pH < 0.10
  - Respiratory rate < 35 breaths/minute
  - Heart rate < 140 or increased < 20% from baseline
  - Systolic blood pressure > 80-160 mmHg or change < 20% from baseline
- *Subjective criteria*
  - Resolution of disease acute phase

- Physician believes discontinuation possible
- Adequate cough / ability to protect airway and clear secretions
- No signs of increased work of breathing, including thoracoabdominal paradox or excessive use of accessory respiratory muscles
- No other signs of distress, such as diaphoresis or agitation

Measurements that have been shown to have statistically significant likelihood ratios to predict the outcome of a ventilator discontinuation effort:

*Measured on ventilator:*

- VE 10-15 L/min
- NIF -20 to -30 cmH2O
- PImax -15 to -30 cm H2O
- P0.1/PImax 0.30
- CROP score 13 [index including compliance, rate, oxygenation, and pressure]

*Measured during a brief period of spontaneous breathing*

- RR 33-38 breaths/minute or less
- VT 4-6 mL/kg
- F/VT ratio 60-105/L (i.e., RSBI, rapid shallow breathing index) ***(Matic, 2004)***.
- Weaning Index: The frequency-to-tidal volume ratio (f/VT) or rapid shallow breathing index (RSBI) is a good predictor of weaning success if the value is low, but not when the value approximates 105. Because of the aforementioned, 2 corrective factors to the RSBI were added. The first one was elastance index (EI = peak pressure/NIF) and the second one, the ventilatory demand index (VDI = minute ventilation/10). The result of the product of the RSBI ×EI ×VDI was called the weaning index (WI).The WI is a simple and reproducible parameter that integrates breathing pattern, compliance, inspiratory muscle strength, and ventilatory demand and is the most accurate predictor of weaning success***(Armando et al., 2012).***

Some clinicians recommend alternating trials of spontaneous breathing, lasting from a few minutes to a few hours, with full ventilatory support. The trial of spontaneous breathing may last up to 2 hours; although recent evidence suggests that a 30-minute trial is equally as efficacious in predicting successful weaning ***(Matic, 2004)***.

After passing the previous criteria, the decision of extubation comes. It rests on several criteria:

- Glasgow score ≥ 8.
- Ability to clear airway secretions and to expectorate.
- Effective glottis function.
- Absence of laryngeal edema.

Finally, four factors may lead to failure of identifying weaning readiness and prolong the weaning process:

1) Failure to repeatedly screen patients on a routine basis, due to the absence of a standardized weaning procedure.
2) Excessive ventilatory assistance, leading the healthcare staff to under estimate the patient's ability to breathe spontaneously.
3) Organizational problems related to shortage of physicians and nurses.
4) Over sedation ***(Girard et al, 2008).***

Other methods of weaning include gradual reduction in the number of SIMV breaths, which slowly shifts the work of breathing to the patient. A patient may also be gradually removed from PSV; pressure support levels must reach 5 to 7 cm $H_2O$ prior to extubation. In many patients, clinicians combine SIMV with PSV.

When weaning is considered in this population, the number of mandatory SIMV breaths is first decreased to zero and then the amount of pressure support is lowered to between 5 and 7 cm $H_2O$. If the patient tolerates a period of breathing at this level of support, extubation is attempted ***(Wilson, 2009)***.

Trials comparing different modes of weaning have produced conflicting results; Therefore the optimal weaning technique remains unknown. Regardless of method, weaning should be stopped and mechanical ventilatory support should be restarted if signs of fatigue, distress, respiratory acidosis, or hypoxemia occur. After extubation, patients should be closely monitored for the development of respiratory distress, fatigue, or stridor.

# ICU SCORING SYSTEM

There are several scoring systems in intensive care units (ICUs) today. The following scoring systems are for adult ICU.

## *Adult scoring systems*

**General scores**

- ASA Physical Status Classification System
- SAPS II (Simplified Acute Physiology Status): expanded and predicted mortality
- APACHE II and predicted mortality
- SOFA (Sequential Organ Failure Assessment)
- MODS (Multiple Organ Dysfunction Score)
- ODIN (Organ Dysfunctions and / or Infection)
- MPM (Mortality Probability Model)
    - on admission
    - 24 hours
    - 48 hours
- MPM II (Mortality Probability Model)
    - on admission
    - 24 h, 48 h, 72 h
- LODS (Logistic Organ Dysfunction System)
- TRIOS (Three days Recalibrated ICU Outcome Score) ***(Jones, 2009)***.

**Specialized and Surgical Intensive Care - Preoperative evaluation**

- Thoraco score (thoracic surgery)
- Lung Resection Score (thoracic surgery)
- EUROSCORE (cardiac surgery)
- ONTARIO (cardiac surgery)
- Parsonnet score (cardiac surgery)
- System 97 score (cardiac surgery)
- QMMI score (coronary surgery)
- Early mortality risk in redo-coronary artery surgery
- MPM for cancer patients
- POSSUM (Physiologic and Operative Severity Score for the enumeration of Mortality and Morbidity) (surgery, any)
- Portsmouth POSSUM (surgery, any)

- IRISS score : graft failure after lung transplantation
- Glasgow Coma Score

**Trauma scores**

- ISS (Injury Severity Score), RTS (Revised Trauma Score), TRISS (Trauma Injury Severity Score)
- ASCOT (A Severity Characterization Of Trauma)
- 24 h - ICU Trauma Score
- TISS (Therapeutic Intervention Scoring System)
- TISS-28 : simplified TISS

The most important for the study done were SOFA and APACHE II scoring systems

## *SOFA scoring system*

The Sequential Organ Failure Assessment (SOFA) Score has been developed by European Society of Critical Care Medicine (ESCCM), in 1994, as a system for measuring the status of the patient in the ICU. It basically evaluated the six different organ systems separately. Different variables and parameters are included in each of the organ system and a definite score is given to that state varying from0 - 4, all of which is later added to calculate the SOFA score, (out of a maximum of 24). The score increases as the organ system functioning worsens. Thus assessment of individual organ dysfunction or failure can be done along with evaluation of patient as a whole. SOFA score can be used to evaluate all patients in the ICU ***(Acharya et al., 2007)*** (Table 1).

The objective in the development of the SOFA was to create a simple, reliable, and continuous score easily obtained in every institution.

Sequential assessment of organ dysfunction during the first few days of ICU admission is a good indicator of prognosis. Both the mean and highest SOFA scores are particularly useful predictors of outcome. Independent of the initial score, an increase in SOFA score during the first 48 hours in the ICU predicts a mortality rate of at least 50% ***(Jones et al., 2009).***

**Table (1):** The Sequential Organ Failure Assessment Score.

| SOFA score | 0 | 1 | 2 | 3 | 4 |
|---|---|---|---|---|---|
| **Respiration** $PaO_2/FIO_2$ (mm Hg) $SaO_2/FIO_2$ | >400 | <400 221–301 | <300 142–220 | <200 67–141 | <100 <67 |
| **Coagulation** Platelets $10^3/mm^3$ | >150 | <150 | <100 | <50 | <20 |
| **Liver** Bilirubin (mg/dL) | <1.2 | 1.2–1.9 | 2.0–5.9 | 6.0–11.9 | >12.0 |
| **Cardiovascular** Hypotension | No hypotension | MAP <70 | Dopamine </=5 or dobutamine (any) | Dopamine >5 or norepinephrine </=0.1 | Dopamine >15 or norepinephrine >0.1 |
| **CNS** Glasgow Coma Score | 15 | 13–14 | 10–12 | 6–9 | <6 |
| **Renal** Creatinine (mg/dL) or urine output (mL/d) | <1.2 | 1.2–1.9 | 2.0–3.4 | 3.5–4.9 or <500 | >5.0 or <200 |

MAP: mean arterial pressure.

CNS: central nervous system.

SaO2: peripheral arterial oxygen saturation.

$PaO_2/FIO_2$ ratio was used preferentially. If not available, the $SaO_2/FIO_2$ ratio was used.

Vasoactive mediations administered for at least 1 hr. (dopamine and norepinephrine ug/kg/min) ***(Hu et al, 2009).***

- High accuracy:

- The platelet count

- Bilirubin level
- Administered dose of epinephrine
- Administration of dobutamine
- Creatinine level

- Low accuracy:
  - Pao2/Fio2 ratio
  - GCS score
  - urine output

The less accurate variables appear to be those that require judgment on the part of the abstractor (GCS score) and those that require calculations (Pao2/Fio2 ratio and urine output). Many errors were made during the calculation of the Pao2/Fio2 ratio due to incorrect selection of Pao2 or Fio2 values ***(Hu et al., 2009).***

## *APACHE II*

Acute Physiology and Chronic Health Evaluation II (APACHE II) is a severity-of-disease classification system, one of several ICU scoring systems. It is applied within 24 hours of admission of a patient to an intensive care unit (ICU): an integer score from 0 to 71 is computed based on several measurements; higher scores correspond to more severe disease and a higher risk of death ***(Kanus, 1985).***

APACHE II scoring system was developed by Knaus and colleagues in 1985. APACHE II drives an Acute Physiological Score (APS), reflecting patients' physiological status combined with scores for chronic disease state Chronic Health Index (CHI) and Age score to develop APACHE II. The APACHE II score was primarily designed to predict the mortality of patients in ICUs but attempts have been made to apply this scoring system to patients with severe trauma, abdominal complications, COPD and acute pancreatitis. It has been found that this score accurately reflects the degree of physiological derangement and correlates with subsequent clinical course and length of ICU stay. The APACHE II scoring system is widely used in general intensive care units (ICU) for comparative audit, evaluative research, and clinical management of individual patients. The number of acute organ failures has been shown to be an important determinant of prognosis in critically ill patients admitted to an ICU. Critical illness is a dynamic process and therefore the APACHE II scoring system for outcome prediction based on a single time point does not consider changes in patients' clinical status over time and their response to

treatment, which limits its accuracy. Whether the APACHE II scoring system is still applicable to specialized ICUs is not clear, and very few data exist on predicting outcomes for patients in neurologic intensive care unit (N-ICU) ***(Harrison et al, 2006).***

## *Application*

APACHE II was designed to measure the severity of disease for adult patients admitted to Intensive care units. The lower age bound is not specified in the original article, but a good limit is to use Apache II only for patients aged 15 or older.

This scoring system is used in many ways:

- Some procedures and some medicine are only given to patients with a certain APACHE II score.
- APACHE II score can be used to describe the morbidity of a patient when comparing the outcome with other patients.
- Predicted mortalities are averaged for groups of patients in order to specify the group's morbidity.

Even though newer scoring systems, such as SAPS II, have replaced APACHE II in many places, APACHE II continues to be used extensively because so much documentation is based on it.

The system is still the most widely used system for the evaluation of ICU patients due to its simplicity in gathering information through regular tests and examinations and reproducibility. The APACHE II system consists of three basic elements:

1. Patients' acute physiological symptoms are calculated and scored from 12 routine physiological measurements rectal temperature based on Celsius, respiratory rate, heart rate, serum potassium level, serum creatinine level, mean arterial pressure, oxygenation rate with respect to the percent of oxygen uptake and oxygen alveolar-arterial gradient, hematocrit, white blood cells, blood bicarbonate level, level of consciousness using the GCS and arterial PH (Table 3).
2. Patients' age.
3. Previous chronic disorder ***(Minoo et al., 2012).***

The APACHE II score is obtained by summing up the above items. The worst sign and test in the first 24 hours of ICU admission is recorded and calculated in the scoring system. Each variable has a value between zero to four which are respectively the lowest and the worst scores. The APACHE II instrument is commonly used to

predict the mortality rate in intensive care units in the first 24 hours of admission. Several studies have encouraged the application of the mentioned tool for patients' triage in the ICU ***(Hu et al, 2009)*** (Table 2).

Chronic Health Points: If the patient has a history of severe organ system insufficiency or is immunocompromised as defined below, assign points as follows:

- 5 points for nonoperative or emergency postoperative patients
- 2 points for elective postoperative patients

**Definitions:** organ insufficiency or immunocompromised state must have been evident prior to this hospital admission and conform to the following criteria:

- **Liver** – biopsy proven cirrhosis and documented portal hypertension; episodes of past upper gastrointestinal (GI) bleeding attributed to portal hypertension; or prior episodes of hepatic failure/encephalopathy/coma.
- **Cardiovascular** – New York Heart Association Class IV.
- **Respiratory** – Chronic restrictive, obstructive, or vascular disease resulting in severe exercise restriction (i.e., unable to climb stairs or perform household duties; or documented chronic hypoxia, hypercapnia, secondary polycythemia, severe pulmonary hypertension (>40 mmHg), or respirator dependency.
- **Renal** – receiving chronic dialysis.
- **Immunocompromised** – the patient has received therapy that suppresses resistance to infection (e.g., immunosuppression, chemotherapy, radiation, long term or recent high dose steroids, or has a disease that is sufficiently advanced to suppress resistance to infection, e.g., leukemia, lymphoma, AIDS ***(Harrison et al., 2006).***

**Table (2):** APACHE II Score

| Score | Death Rate (%) |
|---|---|
| 0-4 | 4 |
| 5-9 | 8 |
| 10-14 | 15 |
| 15-19 | 25 |
| 20-24 | 40 |
| 25-29 | 55 |
| 30-34 | 75 |
| >34 | 85 |

**Table (3):** APACHE II parameters

| Physiologic Variable | High Abnormal Range | | | | | Low Abnormal Range | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | +4 | +3 | +2 | +1 | 0 | +1 | +2 | +3 | +4 | Points |
| Temperature - rectal (°C) | ≥41° | 39 to 40.9° | | 38.5 to 38.9° | 36 to 38.4° | 34 to 35.9° | 32 to 33.9° | 30 to 31.9° | ≤29.9° | |
| Mean Arterial Pressure - mm Hg | ≥160 | 130 to 159 | 110 to 129 | | 70 to 109 | | 50 to 69 | | ≤49 | |
| Heart Rate (ventricular response) | ≥180 | 140 to 179 | 110 to 139 | | 70 to 109 | | 55 to 69 | 40 to 54 | ≤39 | |
| Respiratory Rate (non-ventilated or ventilated) | ≥50 | 35 to 49 | | 25 to 34 | 12 to 24 | 10 to 11 | 6 to 9 | | ≤5 | |
| Oxygenation: A-aDO2 **or** PaO2 (mm Hg)<br>a. FIO2 ≥0.5 record A-aDO2<br>b. FIO2 <0.5 record PaO2 | ≥500 | 350 to 499 | 200 to 349 | | <200<br><br>PO2>70 | <br><br>PO2 61 to 70 | | <br><br>PO2 55 to 60 | <br><br>PO2<55 | |
| Arterial pH (preferred)<br><br>Serum HCO3 (venous mEq/l) (not preferred, but may use if no ABGs) | ≥7.7<br><br>≥52 | 7.6 to 7.69<br><br>41 to 51.9 | | 7.5 to 7.59<br><br>32 to 40.9 | 7.33 to 7.49<br><br>22 to 31.9 | | 7.25 to 7.32<br><br>18 to 21.9 | 7.15 to 7.24<br><br>15 to 17.9 | <7.15<br><br><15 | |
| Serum Sodium (mEq/l) | ≥180 | 160 to 179 | 155 to 159 | 150 to 154 | 130 to 149 | | 120 to 129 | 111 to 119 | ≤110 | |
| Serum Potassium (mEq/l) | ≥7 | 6 to 6.9 | | 5.5 to 5.9 | 3.5 to 5.4 | 3 to 3.4 | 2.5 to 2.9 | | <2.5 | |
| Serum Creatinine (mg/dl)<br>Double point score for acute renal failure | ≥3.5 | 2 to 3.4 | 1.5 to 1.9 | | 0.6 to 1.4 | | <0.6 | | | |
| Hematocrit (%) | ≥60 | | 50 to 59.9 | 46 to 49.9 | 30 to 45.9 | | 20 to 29.9 | | <20 | |
| White Blood Count (total/mm3) (in 1000s) | ≥40 | | 20 to 39.9 | 15 to 19.9 | 3 to 14.9 | | 1 to 2.9 | | <1 | |
| Glasgow Coma Score (GCS)<br>Score = 15 minus actual GCS | | | | | | | | | | |
| A. Total Acute Physiology Score (sum of 12 above points) | | | | | | | | | | |
| B. Age points (years) ≤44=0; 45 to 54=2; 55 to 64=3; 65 to 74=5; ≥75=6 | | | | | | | | | | |
| C. Chronic Health Points (see below) | | | | | | | | | | |
| Total APACHE II Score (add together the points from A+B+C) | | | | | | | | | | |

***(Harrison et al, 2006)***

# HEAD INJURY

**Definition**

Injury to the head may damage the scalp, skull or brain. The most important consequence of head trauma is traumatic brain injury. Head injury may occur either as a closed head injury, such as the head hitting a car's windshield, or as a penetrating head injury, as when a bullet pierces the skull. Both may cause damage that ranges from mild to profound. Very severe injury can be fatal because of profound brain damage ***(McCance et al., 2002).***

**Description**

External trauma to the head is capable of damaging the brain, even if there is no external evidence of damage. More serious injuries can cause skull fracture, blood clots between the skull and the brain, or bruising and tearing of the brain tissue itself.

Injuries to the head can be caused by traffic accidents, sports injuries, falls, workplace accidents, assaults, or bullets. Most people have had some type of head injury at least once in their lives, but rarely do they require a hospital visit.

However, each year about two million people suffer from a more serious head injury, and up to 750,000 of them are severe enough to require hospitalization. Brain injury is most likely to occur in males between ages 15 and 24, usually as a result of car and motorcycle accidents. About 70% of all accidental deaths are due to head injuries, as are most of the disabilities that occur after trauma ***(Bader et al., 2004).***

A person who has had a head injury and who is experiencing the following symptoms should seek medical care immediately:

- serious bleeding from the head or face
- loss of consciousness, however brief
- confusion and lethargy
- lack of pulse or breathing
- clear fluid drainage from the nose or ear

**Causes and symptoms:**

A head injury may cause damage both from the direct physical injury to the brain and from secondary factors, such as lack of oxygen, brain swelling, and disturbance of blood flow. Both closed and penetrating head injuries can cause swirling movements throughout the brain, tearing nerve fibers and causing widespread bleeding or a blood clot in or around the brain. Swelling may raise pressure within the skull (intracranial pressure) and may block the flow of oxygen to the brain ***(Hickey et al, 2003).***

Head trauma may cause a concussion, in which there is a brief loss of consciousness without visible structural damage to the brain. In addition to loss of consciousness, initial symptoms of brain injury may include:

- memory loss and confusion
- vomiting
- dizziness
- partial paralysis or numbness
- shock
- anxiety

After a head injury, there may be a period of impaired consciousness followed by a period of confusion and impaired memory with disorientation and a breakdown in the ability to store and retrieve new information. Others experience temporary amnesia following head injury that begins with memory loss over a period of weeks, months, or years before the injury (retrograde amnesia). As the patient recovers, memory slowly returns. Post-traumatic amnesia refers to loss of memory for events during and after the accident ***(Littlejohns et al., 2003).***

Epilepsy occurs in 2-5% of those who have had a head injury; it is much more common in people who have had severe or penetrating injuries. Most cases of epilepsy appear right after the accident or within the first year, and become less likely with increased time following the accident ***(McQuillan et al., 2002).***

**Closed head injury**

Closed head injury refers to brain injury without any penetrating injury to the brain. It may be the result of a direct blow to the head; of the moving head being rapidly stopped, such as when a person's head hits a windshield in a car accident; or

by the sudden deceleration of the head without its striking another object. The kind of injury the brain receives in a closed head injury is determined by whether or not the head was unrestrained upon impact and the direction, force, and velocity of the blow. If the head is resting on impact, the maximum damage will be found at the impact site. A moving head will cause a "countercoup injury" where the brain damage occurs on the side opposite the point of impact, as a result of the brain slamming into that side of the skull. A closed head injury also may occur without the head being struck, such as when a person experiences whiplash. This type of injury occurs because the brain is of a different density than the skull, and can be injured when delicate brain tissues hit against the rough, jagged inner surface of the skull ***(Hubble, 2002).***

**Penetrating head injury**

If the skull is fractured, bone fragments may be driven into the brain. Any object that penetrates the skull may implant foreign material and dirt into the brain, leading to an infection.

**Skull fracture**

A skull fracture is a medical emergency that must be treated promptly to prevent possible brain damage. Such an injury may be obvious if blood or bone fragments are visible, but it's possible for a fracture to have occurred without any apparent damage. A skull fracture should be suspected if there is:

- blood or clear fluid leaking from the nose or ears
- unequal pupil size
- bruises or discoloration around the eyes or behind the ears Swelling or depression of part of the head ***(McQuillan et al, 2002).***

**Intracranial hemorrhage**

Bleeding (hemorrhage) inside the skull may accompany a head injury and cause additional damage to the brain. A blood clot (hematoma) may occur if a blood vessel between the skull and the brain ruptures; when the blood leaks out and forms a clot, it can press against brain tissue, causing symptoms from a few hours to a few weeks after the injury. If the clot is located between the bones of the skull and the covering of the brain (Dura), it is called an extradural hematoma. If the clot is between the Dura and the brain tissue itself, the condition is called a subdural hematoma. In other cases, bleeding may occur deeper inside the brain. This condition

is called intracerebral hemorrhage or intracerebral contusion (from the word for bruising).

In any case, if the blood flow is not stopped, it can lead to unconsciousness and death. The symptoms of bleeding within the skull include:

- nausea and vomiting
- headache
- loss of consciousness
- unequal pupil size
- Lethargy ***(Albanese et al, 2003).***

**Post-concussion syndrome**

If the head injury is mild, there may be no symptoms other than a slight headache. There also may be confusion, dizziness, and blurred vision. While the head injury may seem to have been quite mild, in many cases symptoms persist for days or weeks. Up to 60% of patients who sustain a mild brain injury continue to experience a range of symptoms called "post-concussion syndrome," as long as six months or a year after the injury.

The symptoms of post-concussion syndrome can result in a puzzling interplay of behavioral, cognitive, and emotional complaints that can be difficult to diagnose, including:

- headache
- dizziness
- mental confusion
- behavior changes
- memory loss
- cognitive deficits
- depression
- Emotional outbursts ***(McQuillan et al, 2002).***

**Diagnosis**

The extent of damage in a severe head injury can be assessed with computed tomography (CT) scan, magnetic resonance imaging (MRI), positron emission tomography (PET) scans, electroencephalograms (EEG), and routine neurological and neuropsychological evaluations.

Doctors use the GCS to evaluate the extent of brain damage based on observing a patient's ability to open his or her eyes, respond verbally, and respond to stimulation by moving (motor response). Patients can score from three to 15 points on this scale. People who score below eight when they are admitted usually have suffered a severe brain injury and will need rehabilitative therapy as they recover. In general, higher scores on the GCS indicate less severe brain injury and a better prognosis for recovery ***(McCance et al, 2002).***

Patients with a mild head injury who experience symptoms are advised to seek out the care of a specialist; unless a family physician is thoroughly familiar with medical literature in this newly emerging area, experts warn that there is a good chance that patient complaints after a mild head injury will be downplayed or dismissed. In the case of mild head injury or post-concussion syndrome, CT and MRI scans, EEG, and routine neurological evaluations all may be normal because the damage is so subtle. In many cases, these tests can't detect the microscopic damage that occurs when fibers are stretched in a mild, diffuse injury. In this type of injury, the axons lose some of their covering and become less efficient. This mild injury to the white matter reduces the quality of communication between different parts or the brain. A PET scan, which evaluates cerebral blood flow and brain metabolism, may be of help in diagnosing mild head injury.

Patients with continuing symptoms after a mild head injury should call a local chapter of a head-injury foundation that can refer patients to the best nearby expert ***(Bader et al, 2004).***

**Treatment**

If a concussion, bleeding inside the skull or skull fracture is suspected, the patient should be kept quiet in a darkened room, with head and shoulders raised slightly on pillow or blanket.

After initial emergency treatment, a team of specialists may be needed to evaluate and treat the problems that result. A penetrating wound may require surgery. Those with severe injuries or with a deteriorating level of consciousness may be kept hospitalized for observation. If there is bleeding inside the skull, the blood may need to be surgically drained; if a clot has formed, it may need to be removed. Severe skull fractures also require surgery. Supportive care and specific treatments may be

required if the patient experiences further complications. People who experience seizures, for example, may be given anticonvulsant drugs, and people who develop fluid on the brain (hydrocephalus) may have a shunt inserted to drain the fluid.

In the event of long-term disability as a result of head injury, there are a variety of treatment programs available, including long-term rehabilitation, coma treatment centers, transitional living programs, behavior management programs, life-long residential or day treatment programs and independent living programs ***(Hickey, 2003).***

**Prognosis**

Prompt, proper diagnosis and treatment can help alleviate some of the problems after a head injury. However, it usually is difficult to predict the outcome of a brain injury in the first few hours or days; a patient's prognosis may not be known for many months or even years.

The outlook for someone with a minor head injury generally is good, although recovery may be delayed and symptoms such as headache, dizziness, and cognitive problems can persist for up to a year or longer after an accident. This can limit a person's ability to work and cause strain in personal relationships ***(Littlejohns et al, 2003).***

Serious head injuries can be devastating, producing permanent mental and physical disability. Epileptic seizures may occur after a severe head injury, especially a penetrating brain injury, a severe skull fracture, or a serious brain hemorrhage. Recovery from a severe head injury can be very slow, and it may take five years or longer to heal completely. Risk factors associated with an increased likelihood of memory problems or seizures after head injury include age, length and depth of coma, duration of post-traumatic and retrograde amnesia, presence of focal brain injuries, and initial Glasgow Coma Scale score.

As researchers learn more about the long-term effects of head injuries, they have begun to uncover links to later conditions. A 2003 report found that mild brain injury during childhood could speed up expression of schizophrenia in those who were already likely to get the disorder because of genetics. Those with a history of a childhood brain injury, even a minor one, were more likely to get familial schizophrenia than a sibling and to have earlier onset. Another study in 2003 found

that people who had a history of a severe head injury were four times more likely to develop Parkinson's disease than the average population. Those requiring hospitalization for their head injuries were 11 times as likely. The risk did not increase for people receiving mild head injuries ***(Albanese et al., 2003).***

**Prevention**

Many severe head injuries could be prevented by wearing protective helmets during certain sports, or when riding a bike or motorcycle. Seat belts and airbags can prevent many head injuries that result from car accidents. Appropriate protective headgear always should be worn on the job where head injuries are a possibility ***(Hubble, 2002).***

# PULMONARY COMPLICATIONS IN PATIENTS WITH SEVERE BRAIN INJURY

Pulmonary complications are very prevalent in the critically-ill neurological population. Respiratory failure, pneumonia, pleural effusions and empyema, acute lung injury and the acute respiratory distress syndrome (ALI/ARDS), pulmonary edema, and pulmonary embolism (PE) from venous thromboembolism (VTE) are frequently encountered in this patient population. In addition, direct chest trauma and patients with traumatic brain injury (TBI) are not exempt from direct complications such as rib fractures, flail chest, lung contusions, and hemo/pneumothorax.

Unfortunately, the development of these complications extends the patient's need for care in the ICUand prevents early mobilization, and this increases the likelihood of developing secondary disability ***(Pelosi et al, 2011).***Direct brain injury, depressed level of consciousness and inability to protect the airway, disruption of natural defense barriers, decreased mobility, and secondary physiopathologic insults inherent to severe brain injury are the main cause of pulmonary complications in critically-ill neurological patients. The goal in the ICU is to prevent, treat, and optimize hypoxemia and maintain oxygen delivery to limit secondary neurological insults. In the absence of feasible pharmacological agents to target these goals, prevention strategies to minimize pulmonary complications such as use of bedside techniques such as thoracentesis, closed thoracotomies (chest tubes), lung-protective ventilator strategies, bundles for prevention of ventilator associated pneumonias(VAP), and deep venous thrombosis (DVT) prophylaxis are the cornerstone in the prevention and management of pulmonary complications in severe brain injured patients ***(Grady et al., 2012).***

**1-Pulmonary Complications Related to Direct Chest Trauma:**

Patients who sustain TBI are often at risk for the development of other traumatic injuries such as rib fractures, lung contusions, flail chest, and pneumo/hemothorax. The implementation of a routine standardized assessment of the traumatized victim provides a highly sensitive protocol to diagnose these injuries (figure 10). Most of the times patients require mechanical ventilation and pain control but the decision to intubate may require individualization ***(Seder et al, 2009).***

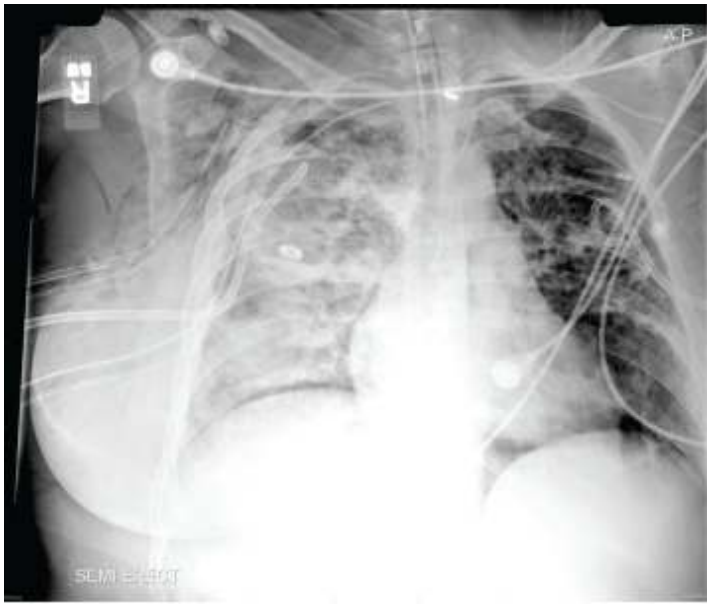


**Figure 10**: Chest X-ray of an ARDS victim who has developed multiple pneumothoraces secondary to a bronchopleural fistula ***(Seder et al, 2009).***

**2-Respiratory Failure and Pneumonia:**

Neurologic related respiratory failure from severe central nervous system dysfunction is one of the most frequent reasons for initiating mechanical ventilation. Among the causes of neurologic dysfunction, structural causes such as ischemic stroke (AIS), hemorrhages intracerebral hemorrhage (ICH) and subarachnoid hemorrhage (SAH)),and TBI carry the worst prognosis and are the greatest challenge to critical care specialists based on the interaction between hypoxemia and secondary neurological insults. Pneumonia is a common complication of severe brain injury and can occur in up to 60% of patients as these patients are prone to aspirate stomach contents. Similarly, ventilator associated pneumonia (VAP) is a preventable secondary consequence of prolonged intubation and mechanical ventilation. Though neurological patients experience more early tracheotomies in general, this practice has not been associated with improved patient outcomes, particularly mortality or onset of VAP. Accumulation of fluid in the pleural space and bacterial infection may result in empyema. Treatment of empyema and complicated pleural effusions requires evacuation of the infected material via chest tube and antibiotic regimen ***(Goldstien et al, 2009).***

**3-ALI and ARDS:**

ALI and the more severe form of lung injury, the ARDS, are a continuum of inflammatory responses following director indirect insults to the lung and clinically recognized by the onset of hypoxemia, reduced pulmonary compliance, and radiographic appearance of bilateral infiltrates (figure 11). The incidence of ALI/ARDS syndrome has been reported in 20–25% of patients with isolated TBI. In patients with SAH an incidence of 20–30%has been reported as well, and in acute ischemic stroke (AIS), a recent epidemiological study reported that the cumulative incidence of ARDS from 1994 to 2008 was4%. In all reports, the mortality and outcomes are substantially the worst ***(Kahn et al., 2006).***

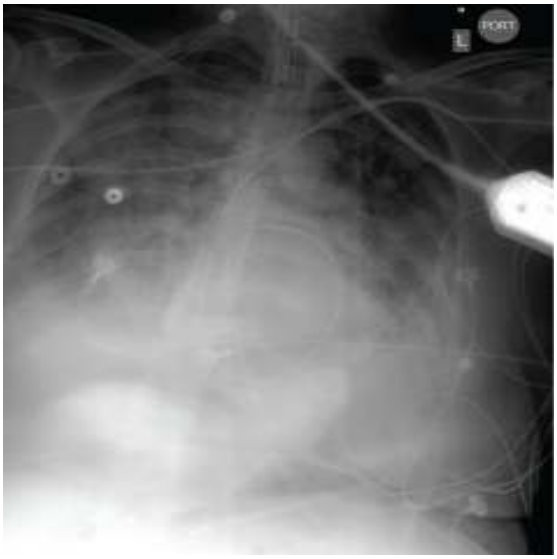


**Figure 11**: Chest X-ray of an ARDS Victim that suffered a grade 4SAH ***(Kahn et al, 2006)***

**4-Pulmonary Edema in the SAH Population:**

Symptomatic cerebral vasospasm and delayed cerebral ischemia continue to be a major etiology for significant morbidity for patients suffering from acute, aneurysmal SAH. Triple H therapy, which consists of hypertension, haemodilution and hypervolemic therapy, has been a mainstay of the medical therapy for treating symptomatic vasospasm for the past few decades. Each component of this therapy is geared towards augmenting cerebral blood flow (CBF) and perfusion pressure of the brain. Despite a paucity of evidence for indications, triple therapy has been widely used around the world as both prophylaxis and treatment for cerebral vasospasm. Regardless of whether it is used as a prophylaxis or treatment for active vasospasm, hypervolemia and haemodilution frequently lead to medical complications most often as pulmonary edema and anemia, which could be associated with worst outcomes in SAH patients (figure 12).The debate regarding the use of triple H therapy and fine balance between maximizing CBF in the setting of pulmonary edema may continue, and the answer may be similar to many other controversial topics: individualized, case-by-case decisions. In any event, the principle of adequate gas exchange needs to be conveyed and achieved in order to successfully optimize brain oxygenation. Current guidelines for the management of SAH support the maintenance of euvolemia rather than hypervolemia ***(Ghosh et al., 2012).***

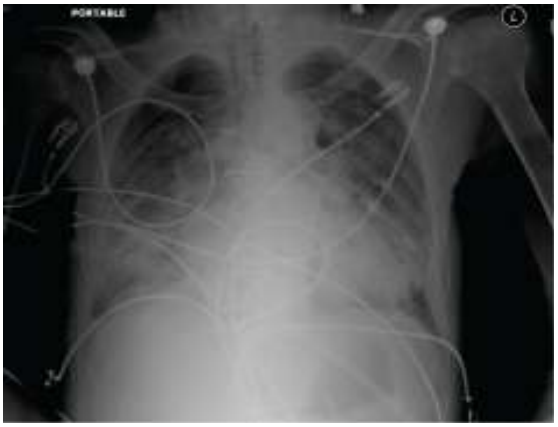


**Figure 12**: Pulmonary edema from volume overload as complication of Triple H Therapy in a patient with Grade 4 SAH. Note distended pulmonary arteries and prominent cardiac silhouette ***(Ghosh et al, 2012)***

**5-Neurogenic Pulmonary Edema:**

Neurogenic pulmonary edema (NPE) has been reported with a number of proposed mechanisms. While the exact locations and circuits involved in the central nervous system (CNS) have not been clearly identified, this uncommon but potentially life threatening condition may occur in the setting of acute, severe brain injuries including traumatic brain injury, intracerebral hemorrhage (ICH), and even in seizures. Sudden rise in intracranial pressure (ICP) such as in SAH or ICH, hypothalamic involvement, rapidly occurring sympathetic surge, increased systemic vascular resistance (SVR) have all been implicated in pathophysiology. Elevated tone of venous circulation results in more venous return. Increase in hydrostatic pressure in the pulmonary vasculature may lead to interstitial edema formation ***(Mascia et al., 2009).***

**6-Pulmonary Embolism:**

Venous thromboembolism (VTE) is a frequent and serious disease that encompasses both DVT and PE. The epidemiology of deep venous thrombosis and pulmonary embolism in severe brain injured patients varies according to the population studied, injury severity, associated comorbidities and injuries, and the diagnostic methods. In trauma patients, the prevalence of DVT is 18– 60% and that of PE is 4–22%. Studies in cohorts of ICH patients demonstrate a prevalence of 2% for PE and1% for DVT. In the SAH population, the prevalence of PE is <1% and of DVT is 5–7%. The effects of VTE may be detrimental for the critically-ill neurological patient, leading to postphlebitic syndrome, recurrent VTE, and potentially PE with a

mortality rate of 9–50%. Clinical diagnosis of VTE is very difficult, and the sensitivity and specificity of clinical exam are very poor. Therefore, studying patients at higher risk or with higher prevalence of risk factors for the development of VTE requires use of invasive and noninvasive testing (figure 13). Even with the use of pneumatic compression devices, the higher incidence of DVT makes prophylactic heparin therapy desirable. Mechanical devices for DVT prophylaxis are considered to be a standard of care. As opposed to pharmacological prophylaxis, mechanical devices may minimize hemorrhagic complications but may not sufficiently reduce the VTE rates. In general, severe brain injured patients benefit from early use of pharmacological prophylaxis for VTE ***(Kurtz et al., 2010).***

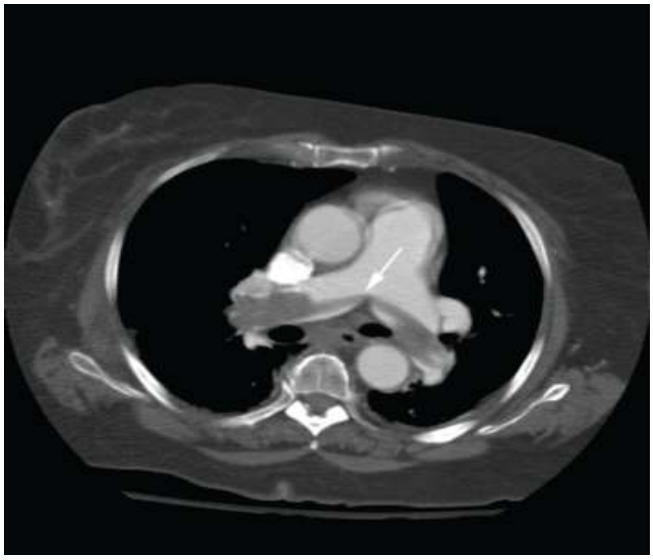


**Figure 13: Chest CT scan shows** Saddle Pulmonary Embolism (white arrow) in a Patient with SAH ***(Kurtz et al, 2010)***

# MECHANICAL VENTILATION AND THE HEAD INJURY

Ventilatory management of a head injured patient is challenging. Injury to the brain initiates an inflammatory cascade that may result in secondary brain injury and extra cranial organ dysfunction. The lung is often the most compromised in this process, and at multiple stages of brain injury. Lung pathology can be part of the initial injury process, or result from sequel of the brain injury and critical care course. Principles of lung protection and brain-directed therapies are often in direct conflict. There are limited randomized controlled trials from which clinicians can draw conclusions regarding management of this controversial cohort of patients. Physiological interactions between the brain and lung should be clearly understood. Injurious ventilation should be avoided. Secondary brain injury should be prevented. Risk factors for this must be identified early and treated promptly. Hypoxemia should be avoided. Arterial CO2 tension should be managed. Hyperventilation should be reserved for intractable intracranial hypertension. There is no role for prophylactic hyperventilation as primary therapy. Hypocarbia can precipitate cerebral ischemia. Novel ventilatory strategies are in the infancy stages. By using these therapeutic modalities, more positive outcomes are hoped for. Traumatic brain injuries are associated with significant morbidity and mortality. Management of a brain-injured patient is complicated by the high incidence of extra cerebral complications in particular pulmonary, making ventilator management extremely challenging. Secondary brain injury, from hypoxemia/hypoxia and hypercarbia, requires prevention and prompt management. This needs to be balanced with lung protective strategies ***(Pelosi et al, 2005).***

**Indications for endotracheal intubation and mechanical ventilation in head injury**

Twenty per cent of brain-injured patients require endotracheal intubation and ventilation.

*Neurologic indications:*

• Altered level of consciousness/airway protection.

• Brainstem dysfunction.

• Intracranial hypertension.

• Anticipated neurologic deterioration.

*Respiratory indications:*

- Hypoxemic respiratory failure may be due to aspiration, pneumonia, atelectasis and pulmonary embolism. Twenty to 45% of patients with traumatic brain injury and subarachnoid hemorrhage develop VAP.
- Acute lung injury/acute respiratory distress syndrome (ALI/ARDS). This presents in 10-30%of patients with brain injuries and aneurysmal subarachnoid hemorrhage. ALI/ARDS is an independent predictor of poor outcome in the setting of brain injury. Distribution is bimodal in ALI/ARDS. Early ALI/ARDS occurs at day two to three and late at day seven to eight post initiation of mechanical ventilation ***(Stevens et al., 2008).***
- Neurogenic pulmonary edema. Mechanisms for this are not clearly understood. Two theories predominate, the hydrostatic and capillary leak theories. The hydrostatic theory postulates that, at the time of cerebral injury, there is a sudden massive adrenergic surge which induces intense pulmonary vasoconstriction. This is followed by capillary vasoconstriction and capillary leak resulting in pulmonary edema. This has also been termed the "blast injury theory". The second theory is the capillary leak theory which postulates pulmonary edema formation on the basis of inflammatory injury. Acute brain injury, contrary to what was previously thought, is a major producer of pro-inflammatory cytokines. This leads to a double-hit effect of secondary brain injury and extra cerebral organ injury and dysfunction ***(Nyquist et al, 2008).***

**Intracranial physiology and mechanical ventilation**

The goals of positive-pressure ventilation (PPV) in brain-injured patients are primarily aimed at improving oxygenation and controlling arterialCO2 tension to minimize intracranial hypertension. PPV increases FRC by improving alveolar recruitment, thus optimizing oxygenation. Increased intrathoracic pressure (ITP) increases intracranial pressure (ICP) via these mechanisms:

- Direct transmission of ITP to the intracranial cavity via the neck.
- Increased ITP decreases venous return to the right atrium, and increases jugular venous pressure, thereby increasing cerebral blood volume (CBV) and ICP.
- Decreased venous return decreases cardiac output and MAP. This results in decreased cerebral perfusion pressure (CPP) leading to compensatory cerebral vasodilation, increased CBV and potentially increased ICP, if cerebral autoregulation is impaired ***(Lapinsky et al., 2009).***

**Mechanical ventilatory strategies: conventional ventilation**

Current practice guidelines for ventilator management advocate protective lung strategies to prevent volutrauma, barotrauma, atelectrauma and biotrauma. The principles are to use low tidal volumes (TV) (5-6 ml/kg ideal body weight),maintenance of low mean airway pressures≤ 30 cmH2O, judicious use of positive end-expiratory pressure (PEEP) with Δ pressure ≤ 18 cmH2O, higher respiratory rates and permissive hypercapnia. This is in direct conflict with the previous "brain directed" ventilatory strategies that used TV of 10 ml/kg, highFiO2 and low PEEP or zero end-expiratory pressure. There is proven mortality benefit with the use of low TV, but permissive hypercapnia may precipitate intracranial hypertension. Animal studies indicate a higher incidence of severe pulmonary edema and hemorrhage after exposure to injurious ventilation in the presence of brain trauma. High TV independently predicts ALI/ARDS and poor outcome in brain injury.

Mechanical ventilation predisposes to potentially significant hemodynamic fluctuations. These may be detrimental in brain injury due to impaired autoregulation, rendering the brain extremely vulnerable to CPP fluctuations. Prompt initial intravascular expansion and vasopressor initiation may be necessary ***(Huynh et al, 2002).***

**The role of PEEP**

Head injured patients are at high risk for associated pulmonary pathology, as part of the initial injury (pulmonary contusion, hemopneumothorax) or as sequel of the brain injury (secondary pulmonary complications). Maintenance of adequate brain tissue oxygenation is paramount for a favorable outcome. PEEP improves oxygenation by recruitment of atelectatic alveolar units, improving FRC and preventing atelectrauma. However, it may have detrimental neurologic effects in certain clinical circumstances. In normal pulmonary compliance; PEEP is associated with increased ITP, decreased right a trial volume, decreased MAP and thus compromised CPP. This situation is not similar to non-compliant lungs, where there is a comparatively low ITP transmission to the cranium, therefore lesser effects on cerebral blood flow (CBF) and ICP. CPP may be indirectly affected by systemic effects of PEEP, but these effects still remain quantitatively modest. PEEP is therefore safe to apply as part of a ventilatory strategy to improve oxygenation. Alveolar over

distension should be avoided and stable hemodynamic parameters should be maintained. Head position also needs attention. At least 30o head elevation promotes intracranial venous drainage via anterior neck veins, as well as the vertebral venous system - which is not majorly affected by ITP. Jugular veins collapse and act as resistors to some of the ITP transmitted. Tight endotracheal tube ties around the neck and extremes of neck rotation should be avoided ***(Davis, 2008).***

**Role of PaCO2 control**

Arterial CO2 tension is a powerful modulator of cerebral vascular caliber, CBF and ICP. The mechanisms are incompletely understood, but CO2 relaxes pial arterioles via interactions between the endothelium, vascular smooth muscle, pericytes, adjacent neurons and glial cells. At physiological PaCO2 ranges, 20-60 mmHg, the relationship between PaCO2 and CBF is linear. Experimental data show that cerebral vessels are sensitive to changes in extracellular pH, rather than a direct response to CO2 or bicarbonate. Therefore increased PaCO2 results in vasodilation, increased CBF, increased CBV, decreased intracranial compliance and increased ICP. The reverse is true for low CO2 tension. This has been the basis for hyperventilation in intracranial hypertension, but cerebral vasoconstriction may precipitate cerebral ischemia as pericontusional areas are sensitive to hyperventilation-induced ischemia.

The Brain Trauma Foundation management guidelines do not recommend hyperventilation for initial management of raised ICP, unless ICP is unresponsive to first-tier therapy or hyperventilation is for very brief periods of time. Maintaining normocarbia is the best practice ***(Lee et al., 2009).***

**Role of brain monitoring during ventilator support in brain injury**

It is prudent to monitor intracranial pressure, CPP, and brain oxygenation during ventilatory support in brain injury. It is pathophysiologically sound to associate high ICP and low CPP with adverse neurologic outcome. However, there is no proven mortality benefit in continuous ICP monitoring. Brain oxygenation may be monitored via jugular venous saturation, near-infrared spectroscopy and micro dialysis catheters. Availability and cost of these devices are limiting factors to their use. Brain tissue metabolism may be monitored with micro dialysis techniques.

**Non-conventional ventilatory strategies**

ICP and brain tissue oxygenation (PbtO2) should ideally be monitored. There are conflicting results on the effects of prone ventilation on ICP and CPP, but there are consistently improved respiratory mechanics and oxygenation. Care should be taken during prone positioning not to dislodge endotracheal tubes and invasive monitors. Pressure-point necrosis should be pre-empted and prevented. There is no mortality benefit to prone positioning ***(Gattinoni et al, 2001).***

*Recruitment maneuvers*

Multiple strategies are used to recruit atelectatic alveoli and improve oxygenation. The use of incremental levels of PEEP and high intermittent tidal volumes require brain physiological monitoring.

*High frequency oscillatory ventilation (HFOV)*

This delivers high mean airway pressure and very small TV of 1-5 ml/kg at a rapid rate. It recruits alveoli, while preventing over distension. Compared to conventional ventilation, HFOV is safe and effective in preventing ventilator-induced lung injury (VILI) and improving oxygenation in severe ARDS. There may be improved intracranial compliance ***(Rienprecht et al., 2003).***

**Liberation from mechanical ventilation in head injury**

The plan to liberate the patient from mechanical ventilation should be made at initiation of ventilation. There needs to be recognition of when mechanical ventilatory support can be reduced and ultimately discontinued. Patients with neurological injury are often difficult to assess, leading to frequent extubation delays. Timely liberation from ventilation has the following advantages:

• Decreased risk of Ventilator Induced Lung Injury (VILI).
• Decreased risk of Ventilator Associated Pneumonia (VAP).
• Decreased airway injury.
• Decreased sedation requirements.
• Decreased delirium.
• Shortened ICU length of stay.

Assessment for extubation readiness can be simplified into three criteria:

• Respiratory criteria.
• Hemodynamic criteria.

• Neurologic criteria including stable neurological status, ICP ≤ 20 mmHg, CPP ≥ 60 mmHg.

Clinicians have to evaluate all risks of premature weaning and extubation. Risks associated with ventilator liberation are:

• Respiratory muscle fatigue.
• Gas exchange failure.
• Loss of airway protection.

There is clear benefit to weaning according to a protocol. There should be frequent assessment of ventilatory support requirement and re-evaluation of factors contributing to ventilator dependence before ventilation is discontinued ***(Salim et al., 2004).***

**Role of tracheostomy**

There is ongoing debate regarding indications and timing for tracheostomy placement. Advantages:

• Decreased risk of self-extubation.
• Decreased sinusitis.
• Decreased airway resistance, dead space and breathing work.
• Better tolerance.
• Less sedative requirements.
• Potentially-reduced duration of mechanical ventilation.

Risks:

• Surgical site infection.
• Airway hemorrhage.
• Pneumothorax.
• Esophageal perforation.

Tracheostomy placement either after 7 days, 10 days or even 14 days according to different institutions leads to earlier liberation from mechanical ventilation, but without any mortality benefit or effect on pulmonary infection rates ***(Combes et al., 2007).***

# PATIENTS AND METHODS

This is a prospective observational study of all mechanically-ventilated head trauma adult patients carried out in a closed 12-bedded general Adult intensive care unit (AICU) in Benha University Hospital in Benha, Egypt. The ICU is attended around the clock by physicians; all are certified and trained in anesthesiology and critical care. The nurse to patient ratio is 1:1 for the entire day; in addition, two senior nurses, and 2 respiratory therapists are present in 2 shifts a day each is 12 hours. Data were collected as a prospective audit, and as no interventions occurred, the local ethics committee did not consider patient relatives consent necessary. The study planned period is 24 months duration from October 2010 to October 2012 and the expected number of the enrolled patients is 120 patients. Patients were classified into three groups according to the method of weaning we used, each included 40 patients.

## *Patients groups:*

**Group I: Synchronized Intermittent Mandatory Ventilation (SIMV)**

In the group that received intermittent mandatory ventilation, the ventilator rate was initially set at half the frequency used during assist–control ventilation; this initial rate was 12.0±2 breaths per minute and mechanical breaths were synchronized with inspiratory effort. We attempted to decrease the ventilator rate, usually by two to four breaths per minute, at least twice a day. The ventilator rate was decreased more rapidly if tolerated by the patient, as reflected by clinical assessment and blood gas monitoring. Patients who tolerated a ventilator rate of five breaths per minute for two hours without signs of distress were extubated. A continuous positive airway pressure of <5 cm of water was permitted.

**Group II: Pressure Support Ventilation (PS)**

In the group that received pressure-support ventilation. Pressure was titrated to achieve a frequency of <25 breaths per minute. Pressure support was initially set at 18.0±6 cm of water, and we attempted to reduce this level of support by 2 to 4 cm of water at least twice a day. The pace was increased if the patient did not have signs of distress. Patients who tolerated pressure support at a setting of 5 cm of water for two hours with no apparent ill effects were extubated. A continuous positive airway pressure of <5 cm of water was permitted.

**Group III: Spontaneous Breathing Trial (SBT)**

In this group, whatever the method of mechanical ventilation was going on, SBT screening is done. If they pass the screening, SBT is done by shifting the ventilator from full support mode to spontaneous breathing with addition of CPAP 5 cm H2O or PEEP 5 cm H2O for not less than 30 minutes and not more than 2 hours. If passed the SBT, the patient can be extubated.

## *Inclusion criteria:*

1) Age between 18 and 50 years.
2) The primary cause of intubation is head trauma.
3) Mechanical ventilation for not less than12 hrs.
5) Absence of tracheotomy.
6) No scheduled surgery in the following 72 hours.

## *Exclusion criteria:*

1) Mechanical ventilation for more than 14 days.
2) Spinal cord injury (SCI) lesions in the cervical region.
3) Elective postoperative neurosurgical patients.
4) No history of bronchial asthma or COPD.
5) Any kind of extubation either planned for less than 48 hours or unplanned extubation which is defined as an accidental or self extubation.
6) Body Mass Index (BMI ≥ 35 kg/m2).
7) Brain dead patients.
8) Multiple organ dysfunction syndrome.
9) Pre-existing decision to limit life support (Do Not Resuscitate, DNR patients).

Unplanned extubation or extubation failure may cause serious complications like difficulty in re-establishing an artificial airway, especially if the patient had already difficult airway, respiratory muscle fatigue and gas exchange failure and that may increase mortality ***(Kress, 2000).***

## *Study outcomes:*

The outcomes of the study could be classified into primary and secondary outcomes:

**The primary outcome:**

Extubation of the patients for a period of at least 48 hours without re intubation.

**The secondary outcomes:**

1. Ventilation days
2. ICU length of stay
3. Hospital length of stay
4. ICU mortality
5. Hospital mortality
6. Unplanned extubation
7. Readmission to the ICU within 48 hours

## *Study protocol:*

We followed the institution ICU policies and procedures, so according to a time-framed daily schedule, the study was implemented.

## *Data collection:*

Age, gender, height, actual and predicted body weight, APACHE II score, maximum SOFA score, and also data about respiration like admission Arterial Blood Gases data like PaCo2, PaO2, PH, PaO2/FiO2 ratio and HCO3and initial ventilation parameters such as mode, rate, FiO2, tidal volume and PEEP, we gave patients numbers without recording the medical record numbers or names as it is considered to be confidential data.

Neurologic assessment included daily determination of the GCS, either by the physician or by a nurse who had been specially trained for this purpose. We arbitrarily designated the verbal component of the GCS as 1 for intubated patients. Intracranial pressure monitoring was performed at the discretion of the treating physician, but it is not the practice in our institution to use ICP monitoring in all comatose patients with acute brain trauma or hemorrhage as a routine. The GCS was used in calculation of APACHE II and SOFA scoring systems and it was incorporated in the criteria of assessment of readiness of weaning to start the process of weaning.

The Acute Physiology and Chronic Health Evaluation II classification system that has been extensively used for predicting the patient mortality in various diseases, (APACHE II) was calculated within the first 24 hours of admission and it included

hemodynamic status, Fio2, PH, HCO3, serum creatinine, sodium, and potassium, Glasgow Coma Score, WBCs, HCT%. It was calculated on a computer based online system (www.mdcalc.com/apache ii score for ICU mortality). It was done only once for each patient (admission score).

The development of the Sequential Organ Failure Assessment **(SOFA)** score was an attempt to objectively and quantitatively describe the degree of organ dysfunction over time and to evaluate morbidity in **ICU** patients.

The **(SOFA)** scoring scheme daily assigns 1 to 4 points to each of the following six organ systems depending on the level of dysfunction: respiratory, circulatory, renal, hematology, hepatic and central nervous system. Since its introduction, the **(SOFA)** score has also been used for predicting mortality, although it was not developed for this purpose ***(Jones et al, 2009).***

SOFA scores were used as well included hemodynamic status and need to inotropic support or vasopressors, serum bilirubin, white cell counts, Po2/FiO2 ratio, Fio2, platelet count, and Glasgow Coma Score, the presence of shock, ventilation data.

SOFA scoring was done for all included patients everyday morning till extubation and mean values were calculated. Then we compared the APACHE II predicted mortality rate with the actual mortality rate, and we saw which was or were the strongest factor (s) or predictors that influence more the reintubation rate in each group and which way of liberation from mechanical ventilation was more suitable according to these factors.

After data collection as mentioned before, patients were connected to mechanical ventilation, one of the full support modes like synchronized intermittent mandatory ventilation (SIMV), volume controlled ventilation (VC), pressure controlled ventilation (PC), and pressure regulated volume controlled ventilation (PRVC) or combined modes like SIMV/PRVC-PS, SIMV/PRVC-PC, SIMV/VC, or SIMV/PC.

The goals of positive-pressure ventilation (PPV) in brain-injured patients are primarily aimed at improving oxygenation and controlling arterial CO2 tension to minimize intracranial hypertension.

According to The Brain Trauma Foundation management guidelines 2007, we did not use the hyperventilation technique and used the best mechanical ventilation parameters for the patient-ventilator synchrony, and in the same time and according to the hospital ICU policy we used the Protective Strategy of Ventilation (PSV) that included high PEEP and low tidal volume. It included VT=6 to 8 ml/kg Predicted Body Weight (PBW), PEEP10-15mmHg, Rate 12-20 breath/minute, FiO2 35%-60%.

Ventilatory goals were PaO2 ≥ 55mmHg, SpO2 ≥ 90% & pH ≥ 7.25.

### *Calculate predicted body weight (PBW):*

- Male = 50 + 0.91 [height (cm) - 152.4].
- Female = 45 + 0.91 [height (cm) - 152.4].

Use these $FiO_2$/PEEP combinations to achieve oxygenation goal.

**Table (4):**FiO2: Inspired oxygen fraction, PEEP: positive end expiratory pressure.

| FiO2 | 0.3 | 0.4 | 0.4 | 0.5 | 0.5 | 0.6 | 0.7 | 0.7 | 0.7 | 0.8 | 0.9 | 0.9 | 0.9 | 1.0 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| PEEP | 5 | 5 | 8 | 8 | 10 | 10 | 10 | 12 | 14 | 14 | 14 | 16 | 18 | 20-24 |

***(Kress, 2000).***

### *Investigations:*

Once patient is admitted to ICU, connected to mechanical ventilation and sedated all the following investigations are done:

Arterial blood gas (ABG).

Complete blood count (CBC).

Random blood sugar (RBS).

Prothrombin time (PT).

Partial thromboplastin time (PTT).

Renal function tests (RFT).

Liver function tests (LFT).

12 leads electrocardiogram (ECG).

Chest X-Ray (CXR).

Any other investigations are done according to any other associated diseases, e.g. CPK, CK, troponin, septic work up etc. Usually CBC, RFT, PT and PTT are repeated daily. CXR is needed only in admission or in specific circumstances such as changing the ETT position, after insertion of central line, check lung fields if there are any clinical changes, or, just before extubation for documentation of healthy lungs. Insertion of an arterial line in the all enrolled patients was mandatory. We always used the radial artery and should be done under complete aseptic technique, confirmed with ABG, and arterial waves on the monitor. No complications recorded encountered.

## *Care of mechanically ventilated patient to prevent Ventilator Associated Pneumonia (VAP) includes:*

1. Positioning 30 to 45 degrees in bed.
2. Chest physiotherapy like postural drainage, vibration/percussion and closed system ETT suction.
3. Bronco-alveolar lavage if needed.
4. Oral suction and hygiene.
5. Proton-pump inhibitors using Pantoprazole 40mg once daily.
6. Daily sedation interruption.
7. Deep venous thrombosis prophylaxis using conventional heparin or unfractionated heparin once daily, sequential compression device (SCD) or thrombo-embolism deterrent (TED) stockings.

## *Sedation:*

Patients were sedated once intubated and mechanically ventilated, and that improves the patient-ventilator synchrony, ETT tolerance, nursing care, and decrease rate of the unplanned extubation.

From that point taking care of sedation and daily interruption of sedation was mandatory. All patients under mechanical ventilation had been kept sedated using one or combination of the following drugs:

**Propofol**

IV infusion: 5-50 mcg/kg/min (20-200 mg/hr.).

**Midazolam**

IV infusion: 2-3 mg/hr.

**Fentanyl**

IV infusion: 50-100 mcg /hr., mainly for pain control.

**Haloperidol**

Either pro re nata (PRN) Dose: 2 to 5mg IV Q 20 minutes PRN ***OR*** Regular intermittent injection: 2.5-10 mg IV Q 6 hr., mainly for anxiety control.

**Muscle Relaxants**

Only for intubation or if there is patient-ventilator asynchrony, muscle relaxants can be used. Patients on muscle relaxants had to be closely monitored by bedside nurses and by Train of Four (TOF) each morning. Daily interruption also of muscle relaxants was mandatory. We used a sedation scale called **Richmond Agitation Sedation Scale (RASS)** ***(Kress, 2000)*****.**

**Table (5):** RASS Score Term Description.

| | |
|---|---|
| +4 | Overtly combative, violent, immediate danger to staff |
| +3 | Very agitated Pulls or removes tube(s) or catheter(s); aggressive |
| +2 | Agitated Frequent non-purposeful movement, fights ventilator |
| +1 | Restless Anxious but movements not aggressive vigorous |
| 0 | Alert and calm |
| -1 | Drowsy Not fully alert, but has sustained awakening (eye-opening/eye contact) to voice (>10 seconds) |
| -2 | Light sedation Briefly awakens with eye contact to voice (<10 seconds) |
| -3 | Moderate sedation Movement or eye opening to voice (but no eye contact) |
| -4 | Deep sedation No response to voice, but movement or eye opening to physical stimulation |
| -5 | Unarousable No response to voice or physical stimulation |

***(Kress, 2000).***

Our target during sedation of mechanically ventilated head injured patients is to keep patients with RASS 0 to -2.

## *Monitoring of the patient under mechanical ventilation:*

- ABG is done every morning, 2 hours after each change in ventilator settings,

and just before and 2 hours after extubation.

- Heart rate, SpO2, blood pressure, and rectal temperature are continuously monitored and recorded hourly.
- If the patient has a cardiac problem, admission ECG is done then every 12 hours as monitoring.
- Fluid balance is calculated every hour, then the net balance every day.
- Blood sugar every 1 hour if patient is under insulin infusion protocol, otherwise measured 8 hourly.
- If patient is on heparin infusion, PT and PTT are measured every 6 hours.
- If patient on continuous renal replacement therapy (CRRT), RFTs are done every 6 hours.
- RASS and GCS are recorded hourly.

### *Monitoring for readiness for starting the process of weaning:*

It was done everyday morning at 7 am by the respiratory therapists for all patients after stopping the sedation and allowing patient to be completely awake or easily arousable (RASS -2).

Daily Interruption of Sedation was done every day for the 3 groups. Then the following screening was done.

It included:

*Neurological status:*

GCS≥8.

ICP < 20 mm Hg (when ICP measured).

CPP ≥ 60 mm Hg (when ICP measured).

*Cardiovascular status:*

Systolic BP > 90 and < 160 mm Hg.

HR > 60 and < 125 beats/min.

No acute dysrhythmia.

No vasoactive agents. (If there was dopamine infusion, it should be not ≥ 0.005mg/kg/min).

*Arterial oxygenation:*

PaO2/FiO2 ≥ 200 mm Hg and/orSpO2 ≥ 95 % on FiO2 ≤ 0.4.

PEEP ≤ 5 cmH2O.

*Spontaneous ventilatory mechanics:*

Rapid Shallow Breathing Index RSBI ≤ 105.

Minimal secretions and effective cough reflex.

*Absence of specific indication for mechanical ventilation:*

Surgery requiring general anesthesia not planned within 72 h.

No deliberate hyperventilation.

Cervical-spine status cleared.

RSBI (f/VT) = Frequency of breathing in 1 min/ averaged tidal Volume in 1 min. This is done on 1 min CPAP of 5 cmH2O.

If the patient fulfilled all the previous criteria, we consider that he or she passed the screening test and that patient can go now for the planned method according to his group. Weaning technique in each group was discussed before.

Successful weaning is defined as a state in which a patient was able to maintain his own breathing for more than 48hours after extubation.

Patients failed the weaning screening or the weaning trial, were reconnected to full ventilatory support for 24 hours before any weaning screening or attempt.

## *Monitoring of weaning failure:*

A weaning failure is defined as failed weaning screening, re-intubation or resumption of ventilatory assist within 48 h or death within 48 h after extubation.

The patient is monitored after extubation continuously for 2 hours for possible re-intubation if showed more than 2 of the following criteria. Otherwise one of the methods of non-invasive ventilation was allowed to be applied such as CPAP 5mmHg or BiPAP 10-12 Ipr /5-8 Epr mmHg to achieve a respiratory rate≤ 25breaths/min and a SaO2≥94% with an FIO2≤0.5, and that were not considered to be weaning failure.

It included:

1. Respiratory rate > 35/min.
2. SpO2 < 90%.
3. Heart rate >140/min.
4. Systolic BP >180mmHg OR <90mmHg.
5. Somnolence, agitation, anxiety.

6. Evident respiratory distress (Diaphoresis, accessory muscle recruitment, thoraco-abdominal paradox) ***(Boles, 2007)***.

# STATISTICAL ANALYSIS

All statistical analyses were conducted using commercially available software (Statistical Package for Social Science 18; SPSS, Inc., Chicago, III). Continuous Variables are presented as medians and interquartile range (25th to 75th percentiles IQR) or means ± SD for continuous variables, as appropriate. Categorical variables are presented as frequency and percentages. Comparisons between the three groups were made using the one-way ANOVA for continuous variables and Chi-squared test for categorical variables. We compared the outcomes between the 3 groups focusing mainly on the primary outcome which is the success rate of weaning. For statically significant one-way ANOVA tests, using Fisher's least significant difference (LSD) were performed. *P*-value was not adjusted for multiple comparisons.Two-tailed tests were used and $P < 0.05$ was considered to be statistically significant in all tests.

# RESULTS

During the study period, 120 patients met the inclusion criteria and they were eligible for the study; all of them were enrolled in the study, 40 patients in each group.

Table 6 shows comparison between the 3 groups regarding the patients demographic data, APACHE II score, maximum SOFA score, and predicted mortality.

The mean age in group I was 30.09±20.93, group II was 36.35±19.55, and group III was 34.72±20.96 with insignificant difference between the 3 groups, P value was 0.12.

The mean height in group I was 163.88±11.26, group II was 165.49±11.53, and group III was 163.88±11.70 with insignificant difference between the 3 groups, P value was 0.77.

The mean weight in group I was 70.96±21.04, group II was 69.80±18.43, and group III was 73.68±22.44 with insignificant difference between the 3 groups, P value was 0.67.

The mean predicted BW in group I was 59.16±11.45, group II was 60.50 ±11.80, and group III was 58.05±11.27 with insignificant difference between the 3 groups, P value was 0.63.

The mean APACHE II in group I was 11.85±6.50, group II was 13.45±7.49, and group III was 11.45±7.32 with insignificant difference between the 3 groups, P value was 0.31.

The mean maximum SOFA score in group I was 9.70±4.33, group II was 8.95±4.07, and group III was 9.52±4.14 with insignificant difference between the 3 groups, P value was 0.10.

The mean predicted mortality in group I was 33.40±19.22, group II was 31.40±20.76, and group III was 32.78±21.40 with insignificant difference between the 3 groups, P value was 0.30.

**Table (6):** Demographic data

| Data | Groups | N | mean | Std. Deviation | Minimum | Maximum | P value |
|---|---|---|---|---|---|---|---|
| **Age in Years** | Group I | 40 | 33.09 | 20.93 | 18 | 50 | 0.12 |
| | Group II | 40 | 36.35 | 19.55 | 18 | 50 | |
| | Group III | 40 | 34.72 | 20.96 | 18 | 50 | |
| | Total | 120 | 46.72 | 21.10 | 18 | 50 | |
| **Height in Cm** | Group I | 40 | 163.88 | 11.26 | 145 | 180 | 0.77 |
| | Group II | 40 | 165.49 | 11.53 | 140 | 182 | |
| | Group III | 40 | 163.88 | 11.70 | 143 | 180 | |
| | Total | 120 | 164.11 | 11.43 | 145 | 183 | |
| **Weight in Kg** | Group I | 40 | 70.96 | 21.04 | 45 | 100 | 0.67 |
| | Group II | 40 | 69.80 | 18.43 | 40 | 105 | |
| | Group III | 40 | 73.78 | 22.44 | 50 | 110 | |
| | Total | 120 | 71.51 | 20.60 | 41 | 103 | |
| **Predicted BW in Kg** | Group I | 40 | 59.16 | 11.45 | 40 | 77 | 0.63 |
| | Group II | 40 | 60.50 | 11.80 | 39 | 78 | |
| | Group III | 40 | 58.05 | 11.27 | 39 | 82 | |
| | Total | 120 | 59.34 | 11.46 | 40 | 81 | |
| **APACHE II score** | Group I | 40 | 11.85 | 6.50 | 9 | 30 | 0.31 |
| | Group II | 40 | 13.45 | 7.49 | 4 | 32 | |
| | Group III | 40 | 11.45 | 7.32 | 6 | 35 | |
| | Total | 120 | 12.22 | 9.90 | 4 | 46 | |
| **Max SOFA score** | Group I | 40 | 9.70 | 4.33 | 1 | 14 | 0.10 |
| | Group II | 40 | 8.95 | 4.07 | 3 | 14 | |
| | Group III | 40 | 9.52 | 4.14 | 3 | 15 | |
| | Total | 120 | 9.16 | 4.30 | 1 | 14 | |
| **Predicted mortality** | Group I | 40 | 33.40 | 19.22 | 10 | 60 | 0.30 |
| | Group II | 40 | 31.40 | 20.76 | ٥ | 55 | |
| | Group III | 40 | 32.78 | 21 .40 | ٧ | 56 | |
| | Total | 120 | 32.85 | 22.42 | 5 | 59 | |

(*P value is significant if ≤ 0.05*)

N: Number.

APACHE II: Acute physiology and chronic health evaluation II.

SOFA: Sequential organ failure assessment.

Std: Standard.

BW: Body weight.

Max: Maximum.

Kg: Kilogram.

Cm: Centimeter.

Table 7 shows the comparison between the 3 groups regarding the gender. In group I males were 31 patients, and females were 9 patients, in group II males were 29 patients, and females were 11 patients, and in group III males were 26 patients, and females were 14 patients. The total number of males in the 3 groups was 86 patients with a percentage of 71.66 % while the total number of females was 34 patients with a percentage of 28.44 % with insignificant difference between the 3 groups, P value was 0.42.

**Table (7):** Gender

| **Weaning groups** | **N** | **Gender** | | **P value** |
|---|---|---|---|---|
| | | **Male** | **Female** | |
| Group I | 40 | 31(77.50%) | 9(22.50%) | 0.42 |
| Group II | 40 | 29(72.50%) | 11(27.50%) | |
| Group III | 40 | 26(65.00%) | 14(35.00%) | |
| Total | 120 | 86(71.66%) | 34(28.44%) | |

(*P value is significant if ≤ 0.05*)

N: Number.

Table 8 shows 3 important secondary outcomes which were ventilation duration, ICU length of stay, and hospital length of stay, all in days.

The mean ventilation duration in group I was 7.89±3.58 days, group II was 8.98±2.96 days, and group III was 9.58±3.47 days with insignificant difference between the 3 groups, P value was o.13.

The mean ICU length of stay in group I was 11.40±5.26, group II was 12.95±4.72, and group III was 13.20±5.52 with insignificant difference between the 3 groups, P value was 0.07.

The mean hospital length of stay in group I was 18.17±8.72, group II was 19.32±7.74, and group III was 20.95±7.31 with insignificant difference between the 3 groups, P value was 0.12.

**Table (8):** secondary outcomes

| Variables | Group | N | Mean | Std.deviation | Minimum | Maximu | P value |
|---|---|---|---|---|---|---|---|
| **Ventilation duration in days** | Group I | 40 | 7.89 | 3.58 | 3 | 14 | 0.13 |
| | Group II | 40 | 8.98 | 2.96 | 2 | 13 | |
| | Group III | 40 | 9.58 | 3.47 | 3 | 14 | |
| | Total | 120 | 8.01 | 3.19 | 2 | 14 | |
| **ICU length of Stayin days** | Group I | 40 | 11.40 | 5.26 | 5 | 18 | 0.07 |
| | Group II | 40 | 12.95 | 4.72 | 6 | 19 | |
| | Group III | 40 | 13.20 | 5.52 | 4 | 21 | |
| | Total | 120 | 11.85 | 8.98 | 3 | 21 | |
| **Hospital length of stay in days** | Group I | 40 | 18.17 | 8.72 | 9 | 23 | 0.12 |
| | Group II | 40 | 19.32 | 7.74 | 10 | 28 | |
| | Group III | 40 | 20.95 | 7.31 | 9 | 26 | |
| | Total | 120 | 19.37 | 8.68 | 8 | 28 | |

(*P value is significant if ≤ 0.05*)

N: Number.

Std.: Standard.

ICU: Intensive Care Unit.

Table 9 shows Fisher´s Least Significant Difference (LSD) test for the dependent variables which were the ventilation duration, ICU length of stay and hospital length of stay, all in days, We found that:

Regarding the ventilation duration in days, there was insignificant difference between group I and group II, P value was 0.09.

There was insignificant difference between group I and group III, P value was 0.08.

There was insignificant difference between group II and group III, P value was 0.76.

Regarding the ICU length of stay in days, there was significant difference between group I and group II, P value was 0.02.

There was insignificant difference between group I and group III, P value was 0.10.

There was insignificant difference between group II and group III, P value was 0.52.

Regarding the hospital length of stay in days, there was insignificant difference between group I and group II, P value was 0.19.

There was significant difference between group I and group III, P value was 0.04.

There was insignificant difference between group II and group III, P value was 0.47.

**Table (9):** Fisher´s Least Significant Difference

| **Variables** | **Groups** | | **P value** |
|---|---|---|---|
| Ventilation duration in Days | Group I | Group II | 0.09 |
| | | Group III | 0.08 |
| | Group II | Group I | 0.09 |
| | | Group III | 0.76 |
| | Group III | Group I | 0.08 |
| | | Group II | 0.76 |
| ICU length of Stay | Group I | Group II | 0.02 |
| | | Group III | 0.10 |
| | Group II | Group I | 0.02 |
| | | Group III | 0.52 |
| | Group III | Group I | 0.10 |
| | | Group II | 0.52 |
| Hospital length of stay | Group I | Group II | 0.19 |
| | | Group III | 0.04 |
| | Group II | Group I | 0.19 |
| | | Group III | 0.47 |
| | Group III | Group I | 0.04 |
| | | Group II | 0.47 |

(*P value is significant if ≤ 0.05*)

ICU: Intensive Care Unit

Table 10 shows the number of patients with unplanned extubation in group I was 3 patients, in group II was 3 patients, and in group III was 5 patients, with total of 11 out of 120 patients, percentage was 9.2 %. There is insignificant difference between the 3 groups, P value was 0.67.

Number of patients with readmission to ICU within 48 hours in group I was 4 patients, in group II was 1 patient, and in group III was 0 patient, with total of 5 out of 120 patients, percentage was 4.2 %. There is insignificant difference between the 3 groups, P value was 0.06.

**Table (10) :** Unplanned extubation and readmission within 48 hours.

| **Unplanned Extubation** | **N** | **Total** | **Percentage %** | **P Value** |
|---|---|---|---|---|
| Group I | 3 | 40 | 7.50 | 0.67 |
| Group II | 3 | 40 | 7.50 | |
| Group III | 5 | 40 | 12.50 | |
| Total | 11 | 120 | 9.20 | |
| Readmission within 48 hours | | | | |
| Group I | 4 | 40 | 10.00 | 0.06 |
| Group II | 1 | 40 | 2.50 | |
| Group III | 0 | 40 | 0.00 | |
| Total | 5 | 120 | 4.00 | |

(*P value is significant if ≤ 0.05*)

N: Number.

Table 11 shows the number of patients died in the ICU in group I was 7 patients, group II was 5 patients, and group III was 3 patients, with total of 15 out of 120 patients, percentage was 12.50 %.there is insignificant difference between the 3 groups, P value was 0.12.

Number of patients died outside the ICU but within the hospital after extubation and discharge to the ward was 7 patients in group I, 6 patients in group II, and 5 patients in group III, with total of 18 out of 120 patients, percentage was 15 %.there is insignificant difference between the 3 groups, P value was 0.20.

**Table (11):** Mortality outcomes

| ICU outcome | Dead | Total | Percentage % | P Value |
|---|---|---|---|---|
| Group I | 7 | 40 | 17.50 | 0.12 |
| Group II | 5 | 40 | 12.50 | |
| Group III | 3 | 40 | 7.50 | |
| Total | 15 | 120 | 12.50 | |
| **Hospital outcome** | | | | |
| Group I | 7 | 40 | 17.50 | 0.20 |
| Group II | 6 | 40 | 15.00 | |
| Group III | 5 | 40 | 12.50 | |
| Total | 18 | 120 | 15.00 | |

(*P value is significant if ≤ 0.05*)

ICU: Intensive care unit.

Table 12 shows the total number of re-intubated patients in the 3 groups was 25 out of 120 patients with a percentage of 20.8 %. They included 11 patients in group I, 9 patients in group II, and 5 patients in group III. There is insignificant difference between the 3 groups, P value was 0.07.

**Table (12):** weaning success (a)

| Reintubation | Number | Total | Percentage % | P Value |
|---|---|---|---|---|
| Group I | 11 | 40 | 27.50 | 0.07 |
| Group II | 9 | 40 | 22.50 | |
| Group III | 5 | 40 | 12.50 | |
| Total | 25 | 120 | 20.80 | |

(*P value is significant if ≤ 0.05*)

Because of this insignificant difference between the 3 groups regarding weaning success as shown in table 12, we did Fisher´s Least Significant Difference (LSD) test to see if there is any significant difference between any one of the 3 groups and any one of the other 2 groups.

Table 13 shows regarding the weaning success, there was insignificant difference between group I and group II, P value was 0.09.

There was significant difference between group I and group III, P value was 0.01.

There was significant difference between group II and group III, P value was 0.04.

**Table (13):** weaning success (b)

| **Variables** | **Groups** | | **P value** |
|---|---|---|---|
| Weaning success | Group I (SIMV) | Group II | 0.09 |
| | | Group III | 0.01 |
| | Group II (PSV) | Group I | 0.09 |
| | | Group III | 0.04 |
| | Group III (SBT) | Group I | 0.01 |
| | | Group II | 0.04 |

Fisher´s Least Significant Difference Test.

(*P value is significant if ≤ 0.05*)

# DISCUSSION

Mechanical ventilation has become a core practice in intensive care units (ICUs) since their inception nearly 4 decades ago. However, it is associated with well documented complications that have a major influence on patient morbidity and mortality. It is therefore imperative to identify the most appropriate method of applying mechanical ventilation and the earliest appropriate opportunity for it to be stopped (termed 'weaning') ***(Mion, 2007).***

Traumatic brain injury (TBI) is worldwide the main cause of morbidity and mortality in individuals less than 45 years old with higher prevalence in the male gender. It takes place in about 40% of victims of trauma, and 20% of them die on the spot or in the first day of admission and 80% in the first seven days after the event.

TBI is a non-degenerative or congenital injury caused by an aggression or started by a process of high energy acceleration or deceleration of the brain inside the cranium which generates an anatomical damage or functional impairment of the scalp, cranium, meninges and encephalus ***(Werner et al, 2007).***

Lowering of the level of consciousness is the main risk factor for broncho aspiration and later admission to the ICU for the purpose of detecting and treating complications of the primary injury and supply a better condition for brain function recovery. Therefore patients with problems related to the central nervous system (CNS) often need ventilation support due to acute respiratory failure (ARF), not always caused by the neurological condition itself, such as decrease of the respiratory drive, but because of lung disease ***(Ferrari, 2006).***

Mechanical ventilation is an essential therapeutic device for patients with tracheal intubation and -severe head injury, since it aims to protect the airway by endo permits sedation and muscle relaxation thus avoiding damages caused by hypoxemia and hypercapnia ***(Braunwald, 2006).***

Clinical data showed worse prognosis in severe TBI, routinely treated with hyperventilation. Hyperventilation becomes appropriate in two situations: (1) treatment of difficult to control ICH; (2) CBF at normal level or high at onset of ICH.

Also when brain deterioration with suspicion of intracranial mass lesion has occurred. Hyperventilation may be needed at times of acute brain deterioration or in long periods of ICH refractory to sedation, paralysis, drainage of cerebrospinal fluid and osmotic diuretics. If, after fluid drainage, ICP remained between 20 to 25 mmHg, hyperventilation must be used in an effort to maintain PaCO2 between 30 to35 mmHg. In presence of ICH refractory to drugs and surgical procedures, PaCO2 <30 mmHg must be maintained with monitoring of jugular venous oxygen saturation (SjO2) and CBF ***(Valadka et al., 2007; Helmy et al., 2007).***

Application of PEEP at 10 and 15 cmH2O levels significantly increased ICP, without significant change in CPP, in patients with acute lung injury (ALI). Increased in levels of PEEP from 0 to 12 cmH2O generated a decrease of MAP in patients with normal compliance and these same values in patients with poor compliance did not bring about significant variations. Therefore, normal respiratory compliance is one of the factors assisting transmission of harmful effects of PEEP to the intracranial system. Careful control of plateau pressure, up to 30 cmH2O must be a rational practice with due surveillance of ICP and CPP when there is elevation of PaCO2. To use or not low levels of PEEP to avoid elevation of ICP is inadequate as it does not correct hypoxemia, which could reduce ICP by better cerebral oxygenation. Effect of PEEP on brain circulation relies on intracranial compliance and on the absolute value of ICP. ICP will not be affected while it remains above CVP generated by PEEP ***(Deem, 2006; Caricato et al, 2005).***

Patients with neurological injury who undergo weaning typically have recovery of ventilatory drive first. This allows ventilation to be changed from controlled to assisted modes. The choice of assisted mode varies, but generally a pressure-support mode with or without permittance of spontaneous breathing (SIMV) is reasonable. This allows a period of gradual recovery of respiratory muscle function. Several predefined weaning protocols have been tried within the medical and surgical ICU settings. There is substantially less evidence supporting the use of these or any other protocol specifically in neurosurgical patients. One recent study investigated the use of a medical ICU protocol in neurosurgical patients and found considerable limitations with its use. Neurosurgical patients as a subpopulation have been intubation -demonstrated to have more difficulty in this period, with higher rates of re being required. Extubation is indicated when spontaneous ventilation is sufficient to

maintain blood gases and patients have the independent ability to protect their airway. Intensive monitoring in this phase is essential ***(McDonagh et al., 2004).***

In a recently published retrospective multicenter cohort study from a prospective compiled and maintained registry, **Pelosi et al**. studied the epidemiology, clinical characteristics, and clinical practices in relation to mechanical ventilation in a cohort of critically-ill neurological patients. Though subarachnoid hemorrhage patients (SAH) were excluded, this study is an excellent description of day-to-day practices across different types of ICUs around the globe. Not surprisingly, neurological patients had lower Glasgow Coma Scale (GCS) on admission, more ICU and ventilator days, had more early tracheostomies, more VAP rates, but more neurological -intubation was similar to those of non-interestingly, the rate of re patients. In this sense, this study provides support that mental status and GCS may not neurological patients and -matter at the time of extubation, as GCS was higher in non intubation was the same ***(Pelosi et al., 2011).***-the rate of re

It should be kept in mind that GCS is only a crude measure of neurologic function. As **King et al** suggested in a comprehensive review of the literature, GCS, inability to follow commands, and airway reflexes maybe independent of each other ***(King et al, 2010).***

Although there are no studies establishing predictive indices for successful extubation in patients with impaired mental status, clinical experience tells us that many patients with profoundly abnormal mental status appear to do well without an artificial airway. In the absence of clear guidelines, clinicians will show substantial variability in their practice and would vary considerably in the timing of the extubation of brain-injured patients once these patients were capable of spontaneous breathing. ***(William et al, 2000).***

The discontinuation or withdrawal process from mechanical ventilation is an important clinical issue. Patients are generally intubated and placed on mechanical ventilators when their own ventilatory and or gas exchange capabilities are outstripped by the demands placed on them from a variety of diseases. Mechanical ventilation also is required when the respiratory drive is incapable of initiating ventilatory activity either because of disease processes or drugs. As the conditions that warranted placing the patient on the ventilator stabilize and begin to resolve,

attention should be placed on removing the ventilator as quickly as possible. Unnecessary delays in this discontinuation process increase the complication rate for mechanical ventilation (*e.g.*, pneumonia or airway trauma) as well as the cost ***(Tobin, 2006).***

Aggressiveness in removing the ventilator, however, must be balanced against the possibility that premature discontinuation may occur. Premature discontinuation establishing artificial -carries its own set of problems, including difficulty in re airways and compromised gas exchange.

There are a number of important issues involved in the management of a mechanically ventilated patient whose disease process has begun to stabilize and/or reverse such that the discontinuation of mechanical ventilation becomes a consideration. First, an understanding of all the reasons that a given patient required a mechanical ventilator is needed (***Tanios, 2006***). Only with this understanding can medical management be optimized. Second, assessment techniques to identify patients who are capable of ventilator discontinuation need to be utilized. Ideal assessment techniques should be able to easily and safely distinguish which patients need prompt discontinuation and which need continued ventilatory support. Third, ventilator management strategies for stable recovering patients who still require some level of ventilatory support need to be employed. These strategies need to minimize both complications and resource consumption (***Girard et al., 2008)*.**

Progress in clarifying the mechanisms that lead to weaning difficulties has markedly improved the management of this crucial phase of artificial ventilation ***(Tobin, 2006).***

Observational studies and randomized trials have established that the time needed for weaning accounts for a substantial proportion of the total time with ventilator assistance, about 40***% (Esteban et al., 2000; Esteban et al., 2002).*** Results of these studies highlight the major impact of the management strategy used for weaning on the outcome of mechanical ventilation. Numerous studies showed that the part of ventilation time devoted to weaning can be significantly shortened and improving ventilator management requires special attention on weaning ***(Esteban et al, 2002)*.**

1990s established that most -The first randomized studies published in the mid patients (usually 60% to 80%) were ready for weaning at the first trial of separation from the ventilator and that the use of a standardized and systematic approach to test readiness for weaning had a strong impact on the duration of mechanical ventilation ***(Ely et al., 1996).*** Furthermore, studies of weaning failure and extubation failure identified specific causes of extubation failure that were not reliably predicted by weaning-readiness tests ***(Epstein and Ciubotaru, 1998).*** A multisociety consensus conference characterized the weaning process as a continuum lasting from intubation until hospital discharge ***(Boles et al., 2007).*** Several international consensus conferences and the publication of expert opinion have failed to produce a set of guidelines for the weaning of patients ***(Boles et al., 2007; Morris and Dracup, 2008).*** Patients are individuals and many have complex comorbidities, consequently there is no universal algorithm, guideline or set of rules that will apply to all. Many weaning trajectories are existing as there are weaning patients (***Egerod, 2003).***

At present, patients are reclassified according to the degree of weaning complexity and some labeled 'weaning failures'. Initial weaning failures occur either because of incomplete resolution of the underlying illness, which necessitated ventilation, or because of the development of a new problem. There are three determinants of weaning. These are the strength of the respiratory muscles, the load applied to these muscles and the central drive. Determinants of weaning can also be used as methods of assessment of the readiness to wean. There are no absolutes in weaning. Much of weaning is trial and error ***(Crocker and Kinnear, 2008).***

Most of the following referenced randomized controlled trials (RCTs) do not include head injured patients, and very few studies address the specific and unique needs of patients with primary neurologic impairment. Head injured patients may "wean off" positive pressure ventilation without difficulty, but they may continue to require an artificial airway secondary to poor mental status or brain stem and or lower cranial nerve deficits. The reasons for failed extubation in this population remain poorly understood. Counterintuitively, a low score on GCS has not been consistently linked with extubation failure and the need for an artificial airway ***(Ko, 2009; Karanjia et al., 2011).***

We applied this accumulated knowledge and evidence in mechanically ventilated critically ill patients with head injury in our study in which we compared the 3 traditional methods of weaning from mechanical ventilation in head injured

patients which are synchronized intermittent mandatory ventilation (SIMV), pressure support ventilation (PSV), and spontaneous breathing trial (SBT), in a prospective design, in a general ICU in order to find out which one of them is the best way of weaning such patients from mechanical ventilation and will be with the highest success rate. Our targeted success of extubation was maintaining patient´s own breathing after extubation for not less than 48 hours.

We put some specific inclusion criteria as described by the Evidence-Based Medicine Task Force of the American College of Chest Physicians, the Society of Critical Care Medicine, and the American Association of Respiratory Care that aimed at keeping patients in the 3 groups having insignificant differences regarding the demographic data, APACHE II score, SOFA score, and hence the predicted mortality rate, as much as we could ***(Ely et al, 2001).***

We found that there was no significant difference between the 3 groups regarding the ventilation duration in days (P value was 0.13), ICU length of stay (P value was 0.07), and hospital length of stay (P value was 0.12). Because we followed an evidence based sedation protocol and we had a well-qualified team of bed side nurses, there was no significant difference between the 3 groups regarding unplanned extubation rate (P value was 0.67). The rate of readmission to ICU after 48 hours from the ICU discharge was of insignificant difference between the 3 groups (P value was 0.06). Also, as expected from the insignificant difference in predicted mortality, the ICU outcome and the hospital outcome, both showed insignificant differences between the 3 groups (P value was 0.12 and 0.20 respectively).

When we found that even the primary outcome which is the reintubation rate had insignificant difference between the 3 groups with a P value of 0.07,we had to do the Fisher´s Least Significant Test to know if there is significant difference between each group and any of the other 2 groups to find out if one is superior to another as a better weaning method and we found that the difference between the SBT and the other 2 groups separately is significant with a P value of 0.01 if compared to SIMV group, and of 0.04 if compared to PS group. But, there was insignificant difference between SIMV and PS groups regarding the weaning success rate (P value was 0.09).

***Esteban et al, (1995)*** faulted the efficacy of all the studies of weaning techniques done before 1980s for having a retrospective design, inappropriate study

populations, and poorly standardized protocols; in addition, most were conducted before the use of pressure support ventilation became widespread **(*Morris and Dracup, 2008)*.**

They compared the 3 methods of weaning which are SIMV, PSV, and SBT.
Like our study did, weaning was considered to have failed if reintubation was necessary within 48 hours after extubation or if extubation was not possible after 14 days from starting ventilation. Weaning was considered to be successful if extubation was achieved within the 14 days period and reintubation was not required within 48 hours of extubation.
According to **Esteban et al** study, insignificant difference between the 3 groups regarding the success rate of extubation was found (P value was 0.54), in addition they found a significant difference when they compared the SBT group to the other 2 groups (to SIMV group, p value was 0.006 and to PS group, P value was 0.04). Comparing the PS group to the SIMV group, insignificant difference was found (P value was 0.32). These results are similar to our study results.

According to the variables used which were similar to the variables used in our study, the only 3 differences between our study and Esteban et al study were in the age, which had significant difference between the 3 groups (P value was 0.02), the ventilation duration in days before starting weaning which was shorter in the SIMV group (P value was 0.005), and the sample size which was more in their study with unequal number of patients between the 3 groups.
Studies of the efficacy of synchronized intermittent mandatory ventilation in weaning have serious limitations. ***Schachter et al., (1981)*** and ***Egerod, (2003)*** compared SIMV to conventional mechanical ventilation and noted no difference between the two techniques in the duration of ventilator support. Their study suffers from a retrospective design, nonuniform study groups (in particular many who required prolonged ventilation for more than 21 days), and inadequate description of the protocol.

***Hastings et al., (1980)*** compared trials of spontaneous breathing with synchronized intermittent mandatory ventilation at a fixed rate (4 breaths per minute) in patients in a stable condition after cardiac surgery. The length of time to extubation was similar in the two groups, approximately 2.6 hours. Their study provides a little insight, however, because 24 hours had already elapsed since the operation and the

patients had a good pulmonary function; thus, little difficulty in weaning was anticipated.

In patients in stable condition who received ventilator support for 3.6 days, ***Tomlinson et al, (1989)*** found that the duration of weaning was similar with spontaneous breathing trials and synchronized intermittent mandatory ventilation approximately 5.6 hours. This study was weighted toward patients who received short term ventilatory support, and two thirds of those weaned within 2 hours were patients who received ventilatory support for less than 72 hours postoperatively (post simple coronary surgeries).

In the previous 3 studies, the duration of weaning was calculated which is not the case in our study, and if compared to Tomlinson et al study, they had a larger sample size (200 patients), unequal number between both groups, and the mean duration of mechanical ventilation was less than 3 days, but in our study the mean duration of mechanical ventilation was 8.01 days.

**Tomlinson et al** conducted his study after several studies had examined the attributes of SBT and SIMV weaning with inconclusive results. Criticism of these studies had included machine variability in relation to the work of breathing, improper application of the 2 weaning methods, and the lack of a heterogeneous study population ***(Tomlinson et al, 1989).***

**Tomlinson et al** concluded that properly executed weaning by either SIMV or SBT can be carried out successfully and rapidly in a similar time frame when simple bed side criteria for weaning are met. In addition, the use of a weaning protocol, after satisfying specific weaning criteria, can facilitate a short wean with a minimum of personnel. ***Brochard et al, (1994)*** compared between the same 3 groups and reported that the PS group had the highest success rate of extubation more than SIMV and SBT groups.

In contrast, we found that weaning with pressure-support ventilation had lower success rate of extubation than SBT group and it was not superior to weaning with SIMV group regarding the success rate. We suspect that the apparent superiority of pressure support in the study by Brochard et al. was due to the constrained manner in which they used the other 2 techniques. Patients had to tolerate an intermittent mandatory ventilation rate of<4 breaths per minute for at least 24 hours before being

extubated. This poses a considerable ventilatory challenge and is not the usual approach to this technique ***(Schachter et al, 1981)***. In contrast, we extubated patients when they tolerated a ventilator rate of five breaths per minute for two hours. In the study by **Brochard et al**, physicians could request up to three trials of spontaneous breathing over a 24-hour period, each lasting 2 hours, before deciding to extubate a patient. Again, this is a considerable ventilatory challenge. We consider the findings of their study and ours to be complementary. Both show that the pace of weaning depends on the manner in which a technique is employed. When synchronized intermittent mandatory ventilation and spontaneous breathing trials are used in a constrained manner, weaning is slower than with pressure-support ventilation. Weaning is expedited when a trial of spontaneous breathing is attempted once a day, and if failed SBT screening, patients should be given a 24 hours rest on full support, as we did in our study.

***Brochard et al, (1994)*** focused more on the total duration of mechanical ventilation as a primary outcome rather than the number of successfully extubated cases in each group as we did in our study.

***Brochard et al, (1994)*** faulted their study and claimed that their results could be affected by many factors, such as, they could not exclude the subjective part of the medical decisions in extubation and there were different criteria of extubation in the 3 groups. They concluded that the way of each technique application could affect any results and better to follow a specific protocol.

***Vitacca et al, (2001)*** compared between PSV and SBT modes as weaning techniques from mechanical ventilation. They found that there was no significant difference between both groups regarding the weaning success rate (P value was 0.12), the ICU and hospital outcomes (P value was 0.09 and 0.15 respectively), the ICU and hospital lengths of stay (P value was 0.30 and 0.20 respectively), and the duration of mechanical ventilation (P value was 0.57).

The study of Vitacca et al had almost the same number of patients (114 patients) and it was similar to our study in other results regarding the patients' characteristics with no significant differences between any of its variables. But that study had some important differences to our study regarding the inclusion criteria which included tracheostomized patients, and ventilation duration

was for at least 15 days, but in our study we excluded the tracheostomized patients and ventilation duration was for not more than 14 days.

**Vitacca et al** concluded in their study that a recent review of the literature of patients in general ICUs (a minority with Chronic Obstructive Pulmonary Disease; COPD) was unable to identify a superior weaning technique among the three most popular modes (SBT, SIMV, or PSV) ***(Esteban et al, 1995; Brochard et al, 1994; Williams, 1980)***. Furthermore recent trials have demonstrated that simply introducing a protocol or guideline for the weaning process leads to a decrease in weaning time and higher success rate independent of the mode used ***(Brochard et al, 1994)***.

***Matic and Majeri-Kogler, (2004)*** compared SBT and PSV as two methods of weaning patients from mechanical ventilation. PSV was the superior method of weaning according to rate of successful extubation, time of weaning from mechanical ventilation, total time of mechanical ventilation, and length of ICU stay (P value was <0.001 for all).

In contrast to our study, the SBT weaning technique was better than the PSV technique, with significant difference between both techniques (P value was 0.04). There was also insignificant difference between the ventilation duration in days and ICU length of stay, and we did not calculate the time of weaning per se.

These differences between both studies could be attributed to some causes such as the different sample size (260 patients in **Matic and Majeri-Kogler** study vs. 120 patients in our study) with unequal number between the groups (110 in SBT group and 150 in PSV group) in contrary to our study which had equal number of patients between the groups.

Superiority of **Matic and Majeri-Kogler** study in ventilation duration means less number of days, which means less probability of complications of mechanical ventilation especially ventilator associated pneumonia and that may be affected the results to a limited degree. They recommended for further studies with larger sample sizes to be done to confirm their results which had small sample size.

There have been a number of randomized controlled trials concerned with the actual method of weaning ***(Tomlinson et al., 1989; Yang and Tobin, 1991; Brochard et al., 1994; Burns et al., 1995, Esteban et al., 1995; Howie, 1999; Burns et al.,***

***2000; Cook et al., 2001).*** Some of them was mentioned before and was compared to our study. These studies had not been consistent in its design with four main methodological differences between them and our study. First, the selection of patients has been inconsistent, including patients who were ventilated for both short term and long term. Moreover, the patient group was not homogeneous and included both those following surgery and those with chronic disease. Second, the definition of successful weaning varied from 2 to 48 h post separation from the ventilator, with some studies including extubation as a criterion. Third, weaning failure was inconsistently defined, and fourth, outcome measures were not consistently applied.

A systematic review of the literature in difficult-to-wean patients ***(Butler et al., 1999)*** was undertaken in order to ascertain which of the three commonly used techniques of weaning (SBT, SIMV, or PSV) leads to the highest proportion of successfully weaned patients in the shortest time. The review demonstrated that there was a lack of randomized, controlled trials designed to determine the most effective technique of patients who were weaned. Furthermore, the manner in which the mode of weaning is applied may have a greater effect on the likelihood of weaning than the mode itself ***(Butler et al., 1999)***.

Many controversial questions remain concerning the best methods for conducting the process of weaning. An International Consensus Conference was held in April 2005 to provide recommendations regarding the management of this process. An 11 member international jury answered these inquiries and they mainly recommended that weaning should be considered as early as possible in patients receiving mechanical ventilation, a majority of patients can be successfully weaned on the first attempt, SBT is the major diagnostic test to determine if patients can be successfully extubated, and they recommended also that specific protocols should be implemented to improve weaning outcome ***(Boles et al., 2007)***.

From the point of view which is the lack of consensus regarding the optimal weaning method, researchers has been prompted to investigate the use of protocols ***(Knebel, 1996; Djunaedi et al., 1997; Blackwood et al., 2004; Tonnelier et al., 2005)*** and the role of the health care professional in weaning ***(Fulbrook et al., 2004)***.

**Namen et al** took the first step in applying a respiratory therapist-driven weaning protocol, incorporating daily screens with SBTs and prompts to caregivers,

in a neurosurgical ICU. All decisions regarding patient care were made by attending neurosurgeons. Namen et al used a randomized controlled study design. It revealed that daily screening (DS) followed by an SBT and extubation readiness prompts to caregivers can reduce the duration of MV, reduce intensive care costs, and is associated with fewer complications than usual. Primary outcomes were the overall duration of MV, LOS in ICU/hospital, and time to successful extubation. Secondary outcomes were the frequency of complications (reintubation, self-extubation, tracheostomy, and MV exceeding 21 days), cost, and mortality ***(Namen et al., 2001).***

The differences between their study and our study were in that they used the reintubation rate as a secondary outcome, and they included tracheostomy, cost and MV for more than 21 days. The most important information needed to support our results was their use for SBT to wean these neurosurgical patients.

***MacIntyre, 2007*** mentioned in his study about (Discontinuing Mechanical Ventilatory Support) that several large clinical trials have clearly demonstrated that many assessment or management strategies can cause considerable undue delay in ventilator withdrawal ***(MacIntyre, 2011; Brochard, 1994).*** Moreover, some trials of protocol driven ventilator discontinuation procedures have clearly demonstrated that traditional "standard care" is often associated with significant delays in ventilator withdrawal ***(Ely et al., 1996; Kollef et al., 1997).***

The advantages to protocols are that a consistent evidence-based approach is applied to all patients and that regular patient assessments are assured. In numerous studies, non-physician-run protocols consistently produce faster ventilator discontinuation times when compared to physician-run "usual care." ***(Randolph et al., 2002).***

***Esteban and Alia, 1998*** mentioned in their review about (clinical management of weaning from mechanical ventilation) that it is surprising that guidelines regarding the optimal approach to the process of weaning are so few at that time ***(Slutsky, 1994).*** Fortunately, several randomized studies carried out in recent years have yielded valuable information in the development of guidelines ***(Ely, 1996; Brochard, 1994***). All have shown that the duration of ventilation and weaning can be reduced by the implementation of specific strategies, such as, using protocols to wean patients instead of the traditional practice based on the personal preferences of physicians, also

clinicians should use the method they feel most comfortable with and individualize the strategy to meet the patient's needs.

***Krishnan et al (2004)*** conducted a prospective controlled trial comparing protocol based weaning to usual physician directed weaning in a closed medical ICU with high physician staffing levels and structured, system-based rounds.

The weaning technique used for both groups was SBT. They found that the only significant difference was regarding the duration of SBT till successful extubation for more than 48 hours with P value of 0.01. All other outcomes, such as, ICU and hospital lengths of stay, and mortality, had insignificant differences.

They concluded that in contrast to the other studies that encourage the implementation of weaning protocols, weaning by nursing and respiratory therapists according to a protocol did not reduce duration of mechanical ventilation, lengths of hospital or ICU stay, or mortality compared with weaning by physicians. They speculate that this lack of benefit may have been due to the high levels of physician staffing in their intensivist-run closed ICU or the use of a template on rounds to promote daily discussion of mechanical ventilation on each patient. Protocols, which are laborious to design and implement, do not necessarily improve patient care and should be tested in the setting in which they are to be applied. The most cost-effective ICU physician staffing level is unknown and will vary among ICUs. However, intensivists inarguably should be attentive to weaning. This attention may be promoted by a weaning protocol, structured rounds, additional staffing, or other tools to ensure that the ability to breathe is recognized promptly.

***Kollef et al. (1997)*** conducted a randomized controlled study comparing protocol directed versus physician directed weaning in two medical and two surgical ICUs in two hospitals. The protocol-directed group was led by nurses and respiratory therapists. The study revealed that nurses and respiratory therapists using protocols weaned patients safely, more quickly and initiated weaning earlier than the physician directed group. There was no blinding to the study and this may have caused a difference, especially in terms of the initiation of weaning. There was no single protocol. Physicians were allowed to draw up their own protocols and these varied between the units.

***Tonnelier et al, (2005)*** compared a prospective protocol led weaning by nurses with a historically matched cohort in a French ICU. It showed that nurse protocol directed weaning reduced duration of mechanical ventilation and length of stay in ICU. Ventilator associated pneumonia, ventilator discontinuation failure rates and ICU mortality were similar in both groups ***(Tonnelier et al., 2005)***.

Arguably, protocols may have merit in institutions where organizational structures and the environment, such as lack of availability of medical staff and critical care nurses educated at postgraduate level, negatively affect the duration of ventilation and progress of weaning. The only identified study of the use of weaning protocols, conducted in Australia, found a prolonged duration of mechanical ventilation with the introduction of weaning guidelines developed by a multidisciplinary team ***(Keogh et al., 2003)***.

Authors attributed the lack of effect on duration of mechanical ventilation to organizational structures and existing nurses' practices that favored rapid decision-making about mechanical ventilation rather than to the protocol itself. This finding is similar to that of ***(Krishnan et al., 2004)***, who directly attributed the lack of effect of a weaning protocol to organizational factors.

Various aspects such as patient communication while being ventilated , patient recollections of stressful experiences while being ventilated, or patients' perceptions of fatigue, all are still under research and their roles in weaning from mechanical ventilation are still under investigations ***(Rotondi et al., 2002)***.

In a Canadian study of 20 patients undergoing ventilation and weaning, it was revealed that patients were actively engaged in a variety of physical, cognitive and emotional activities that contributed to successful weaning. These activities were called patients' work. The authors concluded that patients' work should be understood and supported by clinicians in order to facilitate recovery from mechanical ventilation and weaning ***(Logan and Jenny, 1997)***.

The current protocol of weaning patients from mechanical ventilation mentioned in the review of our study and the current standard mode is the spontaneous breathing trial, and if we look in depth to **Krishnan et al** study we will find a good support of the results of our study which is the method of weaning they used in their study which was SBT, like our study results.

Spontaneous breathing trial approach allows speedier weaning than approaches offering partial ventilatory support like PS and SIMV modes. This approach simplifies management, since a patient's ability to breathe spontaneously without ventilatory support needs to be assessed daily. In contrast, with synchronized intermittent mandatory ventilation and pressure-support ventilation, ventilator settings must be adjusted repeatedly.

An implied goal of the various weaning techniques is to recondition respiratory muscles that may have been weakened during the period of mechanical ventilation. Theoretically, a daily trial of spontaneous breathing and a prolonged period of rest (for 24 hours between failed screening trials) may be the most effective method of eliciting adaptive changes ***(Tomlinson et al., 1989)***. This approach meets the three principal requirements of a conditioning program: overload, specificity, and reversibility ***(Tomlinson et al., 1989)***. During the trial, patients breathe against an elevated intrinsic load, thus satisfying the overload requirement. Specificity is also satisfied, in that the trial is an endurance stimulus and the desired objective is enhanced endurance. Finally, the use of a daily trial prevents regression of the adaptive changes.

**Lazaridis et al, 2012** reviewed 13 different studies from 1994 to 2008. Some of these studies were RCTs and the others were prospective observational and all were about weaning neurosurgical patients from mechanical ventilation. They concluded that our ability to predict “readiness” is poor, and commonly used measures are of questionable value and flawed by inconsistent reports regarding their accuracy. More and better studies are required to evaluate the pathophysiology of extubation failure in brain injured patients, to quantify the risks of reintubation, and to explore better predictors of extubation outcomes in such group of patients ***(Lazaridis et al., 2012).***

Through the results of our study, and all the previous studies and recommendations, we reached to the following recommendations. First, spontaneous breathing trial as a strategy has to be taken in consideration and implemented if choice is made between it and synchronized intermittent mandatory ventilation or pressure support ventilation as methods of weaning patients from mechanical ventilation.

Second, from the insignificant difference in the rate of successful extubation between the previous 3 methods, we recommend to design, follow, and imply a

weaning protocol by a well-educated, highly professional, and multidisciplinary team who has to assure a strong adherence to this protocol, is better than the mode of weaning used.

Third, to respect the patients´ work in the weaning process, and to understand to what extent they have a role in the weaning decision making is very important in the success of weaning the patient from mechanical ventilation.

# CONCLUSION

ICUs vary in their organizational structure and the roles, responsibilities and skills of the various professional groups: medical, nursing and allied health. These differences potentially have a direct effect on the implementation and efficacy of weaning processes.

Comparison of the studies of weaning practices in different international settings is problematic due to socio-political, economic and cultural differences, all of which affect organizational and professional issues in the management of critically ill patients.

The complexity of weaning mandates the use of structures that promote collaborative decision-making by members of the critical care team.

Our study resulted in insignificant difference between 3 methods of weaning head injury patients from mechanical ventilation which are SIMV, PSV, and SBT. Almost all recommendations from the studies of weaning and mechanical ventilation to date are focusing on organizing an international and easily applicable protocol of weaning. Also we prefer through this study to adhere to the SBT as a method and as a protocol of weaning because it is easily applicable even by the respiratory therapists and the nurses and it is also highly predictive of the weaning success.

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# الملخـــص العربي

إن عملية فصل المرضى من على الجهاز التنفسي الصناعي هي من الأمور الطبية المهمة جدا، ويتم وضع المرضى على جهاز التنفس الصناعي عند فشل عملية التنفس وذلك نتيجة عدم كفاءة الرئتين لإمداد الجسم بالأكسجين الازم والكافي للعمليات الحيوية أو فشلها في عملية تبادل الغازات بين الهواء والدم وذلك بسبب العديد من أنواع الأمراض.

والتنفس الصناعي ضروري أيضا عندما يفشل مركز التنفس نتيجة أمراض أو تناول عقاقير معينة، وبمجرد استقرار التنفس وتحسن الحالة الصحية للمريض وبعد معالجة الأسباب التي أدت إلى وضعه على جهاز التنفس الصناعي يبدأ التفكير السريع مباشرة في كيفية فصل المريض من جهاز التنفس الصناعي، ويعد التأخير الغير ضروري في فصل المريض يؤدى إلى العديد من مشاكل التنفس الصناعى مثل الإلتهاب الرئوي والأضرار بالمجرى الهوائي، وفى الوقت ذاته الاستعجال في فصل المريض قد يؤدى إلى ما يسمى بالفصل الغير الناضج أو الغير مكتمل مما يسبب العديد من المشاكل الأخرى الخاصة بهذا النوع من الفصل مثل عدم القدرة على إستعادة المجرى الهوائي الصناعي مع تعطل تبادل الغازات.

وقد تم حساب الوقت اللازم للفصل من جهاز التنفس الصناعي والذي يقضى خلال فترة وضع المريض على الجهاز التنفس الصناعي ووجد أنه بنسبة ٤٢ % من وقت التنفس الصناعي كله بالكامل وقد تعلو هذه النسبة مع بعض أمراض الجهاز التنفسي، وعملية الفصل هي مشتركة بين المريض والجهاز. فالغرض فيها هو تقليل مساعدة الجهاز لعملية التنفس مع زيادة دور المريض بطريقة تدريجية، وبالتالي تنتقل عملية التنفس بالكامل إلى المريض في النهاية.

الطرق الثلاثة العامة لعملية الفصل من على جهاز التنفس الصناعي هي : محاولات التنفس التلقائي، مساعدة الضغط أثناء التنفس، وتزامن التهوية الإلزامية المتقطعة.

ومن الشائع أن مرضى جراحات المخ والأعصاب يحتاجون التنفس الصناعي مع متابعة العلامات الحيوية للجسم وذلك في وحدات العناية المركزة للمخ والأعصاب. والغرض من التنفس الصناعي بعد هذا النوع من العمليات هو تحسين الأكسجين وحماية المجرى الهوائي للبقاء على التنفس.

ومعظم الدراسات المرجعية المذكورة في فصل المرضي من علي جهاز التنفس الصناعي لم تشمل مرض جراحات المخ أو أصابه الدماغ ودراسات قليلة جداً شملت الاحتياجات الخاصة لمرض العجز العصبي والمخي.

إن مرض أصابه الدماغ يمكن فصلها من علي جهاز التنفس الصناعي بدون أي صعوبة ولكن من المحتمل احتياجهم لوجود مجري هوائي صناعي نتيجة عوائق مخية أو عصبية مزمنة نتيجة الأصابة المسببة للتنفس الصناعي.

ومازال أسباب الفشل في تقليل احتياج مثل هؤلاء المرض إلي المجري الهوائي الصناعي غير مفهومة بالكامل ولم يعد هناك ارتباط بين نقص درجة الوعي بمقياس جلاسكو للغيبوبة وبين الفشل من الفصل من علي الجهاز التنفس الصناعي أو الاحتياج للمجري الهوائي الصناعي بعد الفصل.

يمكن عمل محاولات التنفس التلقائية بطرق متعددة وتشمل (التنفس عن طريق أنبوبة علي شكل حرف T – التنفس بضغط منخفض مستمر في المجري الهوائي – دعم التنفس بضغط منخفض الدرجة من الجهاز التنفس الصناعي – ضبط الجهاز علي مساعدة المريض إذا قامة بأي محاولة للتنفس ولكن بدون أعطاء أي ضغط في المجري الهوائي)، ويمكن استخدام طريقة محاولة التنفس التلقائي كطريقة لمعرفة استعداد المريض

لإزالة الأنبوبة الحنجرية أو كطريقة للفصل من الجهاز التنفس الصناعي وذلك بالزيادة التدريجية لمدة كل محاولة.

لقد تم الإثبات بالدراسات العلمية إن نتائج الفصل بالطريقة الثالثة وهي تزامن التهوية الإلزامية المتقطعة أدت إلي أسوأ نتائج.

إن الجيل الجديد من أجهزة التنفس الصناعي تشتمل علي طرق تنفسية حديثة جعلت الفصل أكثر سهولة وتشمل مثلاً كم الهواء الداخل إلي الرئتين، تعوض الأنبوبة ذاتي الحركة (الأتوماتيكي) والمساعدة التنفسية التكيفيه، وبالرغم من سهولة الفصل بهذه الطرق الحديثة فقد ثبت علمياً بأن النتيجة النهائية للفصل لا تختلف كثيراً عن النتيجة النهائية للفصل بالطرق الثلاثة المذكورة في هذه الرسالة.

إن الفصل بطريقة محاولة التنفس التلقائي هو الطريقة الموحدة والنموذجية لاختبار استعداد المريض للفصل وهي مؤشر الأداء الرئيسي للفصل أو العودة للتنفس الصناعي.

ومن مشاكل الطرق الغير منظمة للفصل من جهاز التنفس الصناعي هي إضافة ساعات إن لم يكن أيام التنفس الصناعي والذي يتم تفاديها بإتباع طرق ومنظمة للفصل، والطرق المنظمة للفصل تشمل (فرق طبية – برتوكولات للفصل – دراسات نتائج – إتباع طرق طبية حديثة للفصل)، وهذا قد يحسن النتائج الطبية والاقتصادية.

وفي هذه الدراسة تم دراسة الثلاث طرق الشائعة والتي تم ذكرها وهي محاولات التنفس التلقائي، مساعدة الضغط أثناء التنفس، وتزامن التهوية الإلزامية المتقطعة علي ١٢٠ مريض خلال مدة ٢٤ شهر بدأت فى أكتوبر ٢٠١٠ وانتهت فى أكتوبر ٢٠١٢ وكل طريقة اشتملت علي ٤٠ مريض. المجموعه الأولى تم فصلها باستخدام تزامن التهويه الألزاميه المتقطعه. المجموعه الثانيه تم فصلها باستخدام طريقة مساعدة الضغط اثناء التنفس. والطريقه الثالثه تم فيها استخدام محاولات التنفس التلقائى.

شملت الدراسه مرضى اصابات الدماغ وتم استبعاد مابعد جراحات المخ والاعصاب او مرضى اصابات الجذع الشوكى عند منطقة الرقبه. وتم المقارنه بهدف الحصول على احسن طريقه لفصل مثل هؤلاء المرضى من التنفس الصناعى لمدة لا تقل عن ٤٨ ساعه ومقارنة تاثيرهم على بعض عوامل تقييم الاداء فى اقسام الرعايه المركزة عالميا مثل مدة البقاء فى العنايه والمستشفى ومدة البقاء على التنفس الصناعى ومعدل الوفيات فى العنايه والمستشفى.

وتم اتباع سياسة المستشفى التى تمت فيها الدراسه مع تجميع المعلومات اللازمه ومتابعة المرضى من الناحية التنفسيه والاكلينيكيه والاستعداد للفصل من التنفس الصناعى الى ان تتم عملية الفصل مع مقارنة النتائج بين الثلاث مجموعات مستخدمين احدث الطرق فى التحليل الاحصائى.

بعد مقارنة الثلاث طرق فى فصل مرضى اصابة الدماغ من جهاز التنفس الصناعى وبالرغم من عدم وجود اختلاف كبير فى اوجه المقارنه التى تم ذكرها الا انه نوصى باستخدام طريقة محاولة التنفس التلقائى وهى الطريقه الثالثه. وذالك لانها اسهل فى التطبيق كبروتوكول للفصل وكطريقه للتنبأ بالاستعداد لبداية عملية الفصل وكما انها يمكن تطبيقها بفنيين التنفس الصناعى وبمساعدة التمريض الدرب والمؤهل وذلك كفريق فى العناية المركزة.

# كيفية جعل فصل المريض من التنفس الصناعي أكثر نجاحاً مع اتخاذ عدة عوامل في الأعتبار

رسالة عملية

*مقدمة من*


**الطبيب/ ممدوح الشحات رزق محمد**

***بكالوريوس الطب والجراحة***

***ماجستير التخدير والعناية المركزة***

***جامعة بنها***


**للحصول علي درجة الدكتوراه في التخدير والعناية المركزة**

*تحت إشراف*


**أ.د/ أيهاب أحمد عبد الرحمن**

أستاذ التخدير والعناية المركزة

كلية الطب

جامعة بنها

**أ.د/ محمد يسري عبد المنعم**

أستاذ التخدير والعناية المركزة

كلية الطب

جامعة بنها



**كلية الطب**

**جامعة بنها**

**٢٠٠٩**